In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 19-788V

Filed: June 24, 2025

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TAMI JONES, *Trustee for the heirs of Charley Boon, Deceased,*

Petitioner,

v.

SECRETARY OF HEALTH AND HUMAN SERVICES,

Respondent.

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Randall Knutson, Esq., Knutson & Casey Law Firm, Mankato, MN, for petitioner.
Tyler King, Esq., U.S. Department of Justice, Washington, DC, for respondent.

DECISION[1]

Roth, Special Master:

On May 29, 2019, Charley Boon ("petitioner") filed a timely petition pursuant to the National Vaccine Injury Compensation Program, 42 U.S.C. § 300aa-10 *et seq*.[2] ("Vaccine Act" or "the Program"). Petitioner alleged that he suffered from Guillain-Barré Syndrome ("GBS") as the result of an influenza ("flu") vaccine he received on September 27, 2016. Petition, ECF No. 1. Mr. Boon passed away on November 23, 2019. *See* Pet. Ex. 49.

Upon careful evaluation of all the evidence submitted, I find that petitioner has not provided preponderant evidence that the flu vaccine Mr. Boon received on September 27, 2016 caused and/or contributed to the injury alleged.

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[1] Because this Decision contains a reasoned explanation for the action taken in this case, it must be made publicly accessible and will be posted on the United States Court of Federal Claims' website, and/or at https://www.govinfo.gov/app/collection/uscourts/national/cofc, in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). **This means the Decision will be available to anyone with access to the internet.** In accordance with Vaccine Rule 18(b), the parties have 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. Any changes will appear in the document posted on the website.

[2] National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755. Hereinafter, for ease of citation, all "§" references to the Vaccine Act will be to the pertinent subparagraph of 42 U.S.C. § 300aa (2012).

I. Procedural History

The petition was filed on May 29, 2019. ECF No. 1. On August 19, 2020, petitioner filed a Motion to Substitute Patty Boon, the trustee for the heirs of Charley Boon, as petitioner following Mr. Boon's passing. ECF No. 27-28. After Mrs. Boon passed away in 2024, Tami Jones was substituted as both the trustee and petitioner. ECF No. 67-68.

Between June 14, 2019, and October 1, 2020, petitioner filed medical records and affidavits. Petitioner's Exhibits ("Pet. Ex.") 1-25, 51-54; ECF Nos. 7-9, 29. On June 14, 2019, petitioner filed the expert report of Dr. James Dahlgren, along with medical literature. Pet. Ex. 26-48, ECF Nos. 9, 15-17.

Respondent filed his Rule 4(c) Report on January 25, 2021, recommending against compensation. ECF No. 32.

Petitioner then filed three supplemental reports with accompanying literature from Dr. Dahlgren. Pet. Ex. 55-63, ECF Nos. 34-35; Pet. Ex. 64, ECF No. 40; Pet. Ex. 65-72, ECF No. 49.

Respondent filed expert reports and corresponding literature from Dr. Dara Jamieson and Dr. You-Wen He. Resp. Ex A-B, ECF Nos. 36-38; Resp. Ex. C, ECF No. 46; Resp. Ex. D-E, ECF No. 47.

A status conference was held on April 27, 2022, to discuss the issues in the case, including but not limited to the shortcomings of Dr. Dahlgren's reports and his failure to persuasively address the three prongs of *Althen*, and the onset of petitioner's symptoms more than three months after vaccination. Petitioner was ordered to file a status report advising how she wanted to proceed. ECF No. 50.

In lieu of a status report, petitioner filed a cursory pleading on July 7, 2022, titled "Motion for a Ruling on the Record," which simply asked the Court to decide the case on the record as it stands. ECF No. 52. Petitioner was then ordered to provide a more detailed submission. A six-page memorandum was filed on August 11, 2022. ECF Nos. 53-54.

Respondent filed a response on October 26, 2022, arguing that petitioner had failed to provide preponderant evidence that petitioner's September 27, 2016 flu vaccination caused his alleged injuries. ECF No. 57. Petitioner did not file a reply.

I have determined that the parties have had a full and fair opportunity to present their cases and that it is appropriate to resolve this issue without a hearing. See Vaccine Rule 8(d); Vaccine Rule 3(b)(2); *Kreizenbeck v. Sec'y of Health & Human Servs*., 945 F.3d 1362, 1366 (Fed. Cir. 2020) (noting that "special masters must determine that the record is comprehensive and fully developed before ruling on the record."). Accordingly, this matter is now ripe for resolution.

II. Issues to Be Determined

The issues to be determined herein are whether petitioner suffered from GBS or CIDP, whether a flu vaccine can cause GBS or CIDP, and whether either GBS or CIDP can have an onset of symptoms in excess of three months, or at least 94 days, after receipt of a flu vaccine.

III. Factual Background[3]

A. Medical History Prior to the Influenza Vaccine

Petitioner affirmed and his medical records support a prior medical history that included prostate cancer with prostatectomy, skin cancer, appendectomy, lumbar spine fracture at L5, hypertension, hyperlipidemia, seizure disorder with epilepsy, and arteriosclerotic heart disease with angioplasty. Petition at 1; Pet. Ex. 2; Pet. Ex. 5 at 96. In December 2014, he fell at work and fractured his hip, requiring surgery. *See* Pet. Ex. 5 at 97-107. In June 2015, petitioner saw his primary care physician ("PCP") because his wife was concerned that he "waddles" and "shuffles his feet when walking." The PCP noted that he was "off balance." Pet. Ex. 5 at 94. Petitioner saw his PCP for follow-up in September 2015. He complained that he walked abnormally due to his hip fracture and wanted to know if there was something he could take. *Id*. at 93. The PCP noted extensive medical problems, and that petitioner waddled back and forth and shifted his weight consistent with osteoarthritis from his hip fracture. *Id*. An examination was otherwise normal, and petitioner was noted to be doing well, working in the hardware department of Home Depot and walking five to six miles a day at work. *Id*.

On November 10, 2015, petitioner presented for a flu vaccine. His wife reported concerns for Parkinson's disease and dementia. Pet. Ex. 5 at 92. Petitioner received a flu vaccine without event. The PCP noted that petitioner walked up and down the hall and did not shuffle his feet. His wife was given reassurances regarding his mental status but was "not convinced." *Id*.

In February 2016, petitioner's wife called his PCP with continued concerns that he had dementia that was worsening and requested a referral. *Id*. at 92.

B. Medical History Following the Flu Vaccine

Petitioner received a high dose flu vaccine at a CVS Pharmacy on September 27, 2016. Pet. Ex. 3. He was 69 years old.

Petitioner affirmed that on October 24, 2016, he suffered from "severe flu symptoms" and took over the counter medications including Dayquil Severe and Nyquil for three days. Pet. Ex. 2 at 1. Two months later "at the end of December 2016, I was suffering from numbness in my hands and feet on both sides…" Pet. Ex. 2 at 2.

Petitioner presented to the emergency room ("ER") on December 31, 2016, reporting bilateral numbness in his hands and fingertips for several days that started around his knees and went down to his ankles. His feet and toes were not involved. Pet. Ex. 5 at 41, 142. He reported

[3] All references to "petitioner" throughout this section refer to Mr. Charley Boon.

shuffling his feet for the past two to three years and a fall a week ago. He learned forward when he walked. His wife reported that he was "extremely anxious and nervous" and sometimes wakes up at 2:00 am afraid that he will be late to work at Home Depot at 6:00 am. *Id.* at 41. He reported an episode of double vision on Christmas day that lasted for 15 minutes. He also complained of chills and sweats, but no fever or other symptoms. *Id*. He was admitted for testing, which was negative, and he was discharged in stable condition with instructions to follow up with his PCP. *Id*. at 44.[4]

On January 4, 2017, petitioner presented to the PCP for follow-up. His memory was "a little bit off." He fell that morning, was having trouble walking, and was using a front-wheeled walker. Pet. Ex. 5 at 86. Petitioner had an extensive work-up in the hospital including an MRI of the brain. He had ataxia.[5] His underlying diseases were noted as epilepsy with seizure disorder, arteriosclerotic heart disease with old MI status post angioplasty in 1997, prostate cancer, hip fracture two years ago, and peripheral neuropathy. [6] *Id*.

Petitioner was admitted to the hospital on January 10, 2017, with a sudden onset of severe weakness and numbness in both arms and both legs with increasing fatigue. Pet. Ex. 5 at 87; Pet. Ex. 6 at 2. The differential diagnosis included polyneuropathy and GBS. Pet. Ex. 5 at 91. After extensive testing, it was determined that his symptoms were the result of C5 quadriplegia, and he underwent anterior discectomy and fusion at levels C3-C4 and C4-C5 on January 17, 2017. Petitioner's symptoms were "markedly improved" following surgery. Pet. Ex. 6 at 2, 17-19. However, his condition deteriorated again the next day, but his numbness was not as bad as prior to surgery. *Id*. at 2. He was discharged to a nursing home on January 22, 2017, with diagnoses of C5 level quadriplegia with cervical myelomalacia, memory impairment, and anxiety. *Id*.

Petitioner was reexamined by a neurologist on February 7, 2017. Pet. Ex. 5 at 64. The doctor noted that when he was seen on January 11, 2017, his history was consistent with cervical myelopathy at C4-C5 with no findings of transverse myelitis ("TM"), neuromyelitis optica ("NMO"), WNV poliomyelitis, or atypical extremely rapid motor neuron disease. *Id*. He had surgery with improvement of his lower limb sensory symptoms, but he did not have significant motor improvement. His wife reported that he had been walking abnormally "for over a year or two, scuffing the toes of his feet." *Id*. He was last able to stand and walk in December. He lays in bed most of the day, working with a physical therapist at times. He has fluctuating confusion but is normal at times. No imaging or labs had been done since he was in the hospital. *Id*. The impression was quadriparesis in the setting of cervical myelopathy present for perhaps two weeks prior to diagnosis, with an examination at that time notable for marked upper motor neuron pattern quadriparesis C4/C5 level, and lower limb sensory disturbance. *Id*. at 65. He had no sensory complaints following surgery, but still had quadriparesis. The time course, diagnostics, and clinical examination remained consistent with cervical spine dysfunction. There was concern for possible permanent spinal injury perhaps related to cord infarction due to the little improvement post-

[4] A complete record from this hospitalization was filed as Petitioner's Exhibit 6.

[5] Ataxia refers to the "failure of muscular coordination; irregularity of muscular action." *Ataxia*, DORLAND'S ILLUSTRATED MEDICAL DICTIONARY 168 (33rd ed. 2019) [hereinafter DORLAND'S].

[6] Peripheral neuropathy, also called polyneuropathy, is "neuropathy of several peripheral nerves simultaneously." *Polyneuropathy*, DORLAND'S 1468.

surgery and no significant progression. It was also noted that he "might be exhibiting signs of autonomic dysfunction. *Id*.

Between January 22, 2017 and February 21, 2017, petitioner's weakness in his upper and lower extremities increased. Pet. Ex. 5 at 26, 30. He was readmitted to the hospital on February 21, 2017 for additional testing after he was discharged home against medical advice on February 14, 2017. Pet. Ex. 5 at 26, 30, 140; Pet. Ex. 6 at 294. It was noted that his strength was significantly better after surgery, but he had not maintained that improvement and regressed some. His main complaints before surgery were numbness in his arms and legs which was improved after surgery, but his weakness had increased. Pet. Ex. 6 at 294. He developed neurogenic bladder dysfunction. Pet. Ex. 5 at 26.

Extensive testing had not shed light on a diagnosis other than cervical spine stenosis causing quadriplegia. Pet. Ex. 6 at 296. On re-evaluation by neurology, there were no fasciculations, respiratory or bulbar symptoms. *Id*. at 300. Given his time course, prior diagnostics, and plateau and stabilization of neurologic symptoms, there were no other plausible neurological etiologies. The neurologist remained concerned for possible permanent spinal cord injury from prolonged compression or spinal cord infarct, and postoperative seroma or other phlegmon[7] needed to be ruled out. *Id*. at 300-01. On examination, his upper quadriparesis had worsened a bit since he was examined on February 7, 2017. However, there was no history or diagnostics suggestive of or consistent with NMO, transverse myelitis, or any other neurological conditions. The neurologist felt that empirical IVIG would be worthwhile for "very atypical multifocal motor neuropathy, despite history and exam not necessarily consistent with this." *Id*. at 301-02. He received three days of IVIG without any change. Petitioner developed a pressure ulcer and was discharged to San Diego Rehabilitation on February 24, 2017. *Id*. at 301-02, 320, 331, 426.

Petitioner remained at the rehabilitation facility until March 1, 2017. Pet. Ex. 6 at 426. After neurological reevaluation, repeat IVIG was planned for two weeks later. Prednisone was started, along with Bactrim for a UTI. Due to a change in insurance, his stay at the rehabilitation facility was shortened and his PCP and neurologist changed. Petitioner was discharged home, but it was strongly recommended that he be admitted to another facility for further rehabilitation. *Id*. at 428, 434.

On March 1, 2017, the day of his discharge, petitioner was evaluated by a new PCP. Pet. Ex. 10 at 2. His medical history included partial quadriplegia and abnormal movement of his upper limbs and with shuffling gait for the past two years. *Id*. His wife reported some mild memory deficits. His history over the past two months was also detailed, noting that he was working 40 hours a week at Home Depot three months ago but was now in a wheelchair. *Id*. On physical examination, he had weakness in all four extremities that was more pronounced in the proximal muscles and on the right side of his body. His reflexes could not be elicited, and he had muscle wasting. *Id*. at 3.

Petitioner presented to a new neurologist on March 16, 2017. Pet. Ex. 11 at 2. He was unable to walk, had numbness and tingling in his hands and fingers, and had memory problems. The onset of symptoms was reported as around December 28, 2016, when he developed tingling

[7] Phlegmon is "acute subcutaneous inflammation, sometimes with abscess." *Phlegmon*, DORLAND'S 1413.

in all limbs over two days while off work. He went to the ER on New Years Day, was admitted for testing, and sent home on January 3, 2017. On January 6, 2017, he presented to his doctor and was unable to stand. Throughout that weekend, he became immobile and was admitted to the hospital. *Id*. He underwent extensive testing and spinal surgery, but it was unclear if the surgery helped. He spent several weeks in rehabilitation. After additional testing was done, he was admitted to the hospital again. *Id*. He had been shuffling his feet for two years before, and his wife showed a video taken in April 2016 of him sitting, moving the fingers on both hands and questioned if it was a clue to some neurological condition. He received three days of IVIG in February and started feeling better and getting stronger. He was thinking more clearly now. *Id*. The assessment at this visit was idiopathic peripheral neuropathy. Pet. Ex. 11 at 6. The neurologist wrote that petitioner may have had an episode of GBS, but GBS patients do not need to be retreated unless they show worsening, which typically happens before nadir and petitioner was "well beyond that." *Id*. It was unclear if he could participate in extensive physical rehabilitation. However, he was admitted to rehabilitation from March 21, 2017 through April 15, 2017. He was discharged home in stable condition with a diagnosis of GBS. Pet. Ex. 13 at 1-3.

Petitioner returned to his PCP on April 17, 2017, who noted that he was discharged from acute in-patient rehabilitation due to lack of progression. He reported regaining some strength with improvement to fine motor movement. His goal was to go back to work, but he was tired all the time and did not want to participate in rehabilitation. Pet. Ex. 10 at 6.

Mrs. Boon contacted the neurologist in May 2017 to advise that petitioner was declining and to request another round of IVIG. Pet. Ex. 11 at 7. The neurologist asked if petitioner had a spinal tap done, which would have shown elevated protein on CSF consistent with GBS/CIDP. He also noted that petitioner had no reflexes at his last visit, which could be compatible with GBS/CIDP. *Id*. Mrs. Boon confirmed that petitioner had not had a spinal tap. Petitioner was admitted to the hospital that day for five days of IVIG. Pet. Ex. 11 at 7; Pet. Ex. 14 at 1. He was noted to have increasing weakness with difficulty swallowing upon admission. He improved with five days of IVIG but was not strong enough to go home and was transferred to acute rehabilitation. Pet. Ex. 14 at 1-2. The discharge diagnoses included acute exacerbation of underlying GBS, neurogenic bladder, urinary tract infection, and recent left calf deep vein thrombosis. *Id*. at 1.

Petitioner presented to the PCP on June 5, 2017. He was able to stand from a seated position with assistance and take a few steps with a walker. He could shower with a chair and was receiving PT at home. He was able to feed himself. Pet. Ex. 10 at 14.

At a follow-up neurology appointment on June 9, 2017, petitioner reported he was able to get in and out of the car more easily since the IVIG treatment. Pet. Ex. 11 at 9. His wife reported he only does things if told to and he does not seem to be trying to improve. He fell the day before. He received PT at home three times a week. He was supposed to have EMG/NCS testing while in acute rehabilitation but that was never done. *Id.* On examination, the neurologist noted slow mentation, but petitioner demonstrated "fairly good power" in his upper and lower extremities. Reflexes were hard to elicit. He could stand with assistance and take a few steps with help. *Id*. at 10. He was to be monitored for recurrent or worsening weakness, which may require a five-day course of IVIG. The neurologist noted "[i]f this happens then I would diagnose him with CIDP." Petitioner was taking blood thinners, so a lumbar puncture was never done. *Id*. at 11.

Petitioner relapsed and was diagnosed with CIDP on July 28, 2017. His neurologist ordered IVIG every three weeks six months. Pet. Ex. 15 at 73. In the months that followed, his condition declined, and PT was recommended. Pet. Ex. 11 at 15.

Petitioner returned to the neurologist on October 9, 2017. He fell a month prior when his walker got caught and went to the ER. He was still receiving IVIG, was not sleeping well, and lacked motivation to exercise on his own. His memory was better, but he would probably need assistance throughout life. Pet. Ex. 11 at 17. His diagnosis was CIDP with ongoing IVIG needed. *Id*. at 21.

There was a ten-month gap in the medical records. Petitioner was admitted to the hospital again from August 4, 2018 until August 20, 2018 for congestive heart failure. He underwent coronary artery bypass graft, mitral valve replacement, and left atrial appendage clipping and was discharged without complication. Pet. Ex. 22 at 1-2, 24-25. He was hospitalized again from September 12, 2018 until September 16, 2018 for tachycardia and hypotension. Pet. Ex. 52 at 2159.

On November 14, 2018, Petitioner's IVIG was re-ordered for two days every three weeks for six months. Pet. Ex. 53 at 227.

On April 2, 2019, petitioner was admitted to the hospital for a hip fracture following a fall. Pet. Ex. 53 at 257. He underwent pinning of his right hip and was discharged home on April 5, 2019. *Id*.

Petitioner suffered a CIDP flare on August 22, 2019, and was admitted to the hospital for five days of IVIG. He was discharged to acute rehabilitation on August 27, 2019. Pet. Ex. 53 at 747-48.

On November 15, 2019, petitioner's condition worsened, and he was admitted to the hospital unable to stand or lift his arms. He received five days of IVIG treatment without improvement, and a feeding tube was placed. Pet. Ex. 54 at 216-17. His respiratory status worsened, and he passed away on November 23, 2019. The cause of death was listed as acute respiratory failure, CIDP, severe malnutrition, severe deconditioning, atrial fibrillation, neurogenic bladder, and anemia. Pet. Ex. 49.

IV. Expert Opinions

A. Petitioner's Expert, Dr. James Dahlgren[8]

Dr. Dahlgren provided four reports. Pet. Ex. 26; Pet. Ex. 55; Pet. Ex. 64; Pet. Ex. 65. He initially opined that petitioner developed GBS as a result of the flu vaccine. Pet. Ex. 26 at 12. Later, he opined that petitioner developed GBS and CIDP as a result of the flu vaccine and that

[8] Dr. Dahlgren received his medical degree from University of California, San Francisco, and is board-certified in internal medicine. Pet. Ex. 27. He completed residencies in internal medicine at both Boston Veteran's Hospital in Boston and Cedars-Sinai Medical Center in Los Angeles and a fellowship in infectious diseases at UCLA Medical Center in Los Angeles. Dr. Dahlgren is currently in private practice in internal medicine and occupational and environmental medicine, with a subspecialty in toxicology. *Id*.

both diseases share the same causal mechanism, with chronicity being the main difference. Pet. Ex. 55 at 12-13.

1. Dr. Dahlgren's First Report

In his first report, Dr. Dahlgren summarized petitioner's medical history and his receipt of the high dose Fluzone vaccine followed by the onset of numbness in his hands and feet three months after the vaccine. Pet. Ex. 26. Dr. Dahlgren noted that there is no evidence that petitioner had an acute infection, particularly any diarrheal infection, in the weeks prior to the onset of his GBS. Based on the literature he reviewed, "[t]he mechanism of an acute infection is an immune trigger for GBS, a similar mechanism that is triggered by a vaccination." Pet. Ex. 26 at 2. Therefore, the only plausible causative trigger in this case was the flu vaccine. *Id*.

Dr. Dahlgren asserted that GBS is a documented risk of flu vaccination, and "[t]he mechanism for vaccine induced autoimmunity has been extensively studied and is accepted science." Pet. Ex. 26 at 3. A vaccine stimulates the immune system, causing an autoimmune disease like GBS to occur due to loss of tolerance to normal antigens or antigen mimicry. In GBS, the immune system attacks the nerve cells, causing injury to the myelin sheath covering the nerves. GBS patients usually have sensory and motor dysfunction and can have autonomic neuropathy[9] as well. Petitioner had sensory and motor dysfunction, which is "the most common presentation." *Id*.

Dr. Dahlgren addressed the Vaccine Table's 3-to-42-day period for onset of GBS after flu vaccine and petitioner's onset of GBS three months after his vaccine, arguing that "[t]his 42-day latency is arbitrary and fails to consider all the evidence. There are vaccine induced autoimmune disease cases, which have their onset within 42 days, **but not all.**" Pet. Ex. 26 at 3 (emphasis in original). He cited to literature which he argued showed onset in excess of 42 days: the two tables in *Prestel* showed GBS up to 150 days post-H1N1 and seasonal flu vaccine, and that older individuals are at higher risk of GBS following flu vaccine. Pet. Ex. 26 at 4-5; Pet. Ex. 40.[10] *Soriano* showed onset of autoimmune disease 60 days or more following a flu vaccine, and Dr. Dahlgren referenced a table contained therein discussing the onset of giant cell arteritis and polymyalgia rheumatica within 90 days of a flu vaccine. Pet. Ex. 26 at 5-6; Pet. Ex. 45.[11] *Marks & Halpin* reported onset of symptoms up to ten weeks post vaccination, and *Schonberger* showed GBS cases occurring at 16 weeks following flu vaccination. Pet. Ex. 26 at 6-7; Pet. Ex. 39[12]; Pet. Ex. 43.[13] *Kaplan* used eight weeks as the surveillance period to define the parameters of their study of GBS after flu vaccine. Pet. Ex. 26 at 7; Pet. Ex. 37.[14] *Watad*, a study of cases of autoimmune

[9] Autonomic neuropathy is "any neuropathy of the autonomic nervous system, causing symptoms such as orthostatic hypertension, disordered bowel, bladder, or sexual functions, or abnormal pupillary reflexes; it is a complication of many diseases including Adie syndrome, chronic alcoholism, diabetes mellitus, dysautonomia, and Shy-Drager syndrome." *Autonomic neuropathy*, DORLAND'S 1251.

[10] Jürgen Prestel et al., *Risk of Guillain-Barré syndrome following pandemic influenza A (H1N1) 2009 vaccination in Germany*, 23 PHARMACOEPIDEMIOLOGY & DRUG SAFETY 1192 (2014), filed as "Pet. Ex. 40."

[11] A. Soriano et al., *Giant cell arteritis and polymyalgia rheumatica after influenza vaccination: report of 10 cases and review of the literature*, 21 LUPUS 153 (2012), filed as "Pet. Ex. 45."

[12] James S. Marks & Thomas J. Halpin, *Guillain-Barré Syndrome in Recipients of A/New Jersey Influenza Vaccine*, 243 J. AM. MED. ASS'N 2490 (1980), filed as "Pet. Ex. 39."

[13] Lawrence B. Schonberger et al., *Guillain-Barre Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976-1977*, 110 AM. J. EPIDEMIOLOGY 105 (1979), filed as "Pet. Ex. 43."

[14] Jonathan Kaplan et al., *Guillain-Barré Syndrome in the United States, 1979-1980 and 1980-1981*, 248 J. AM. MED.

diseases triggered by an exposure to an immune triggering event, determined a latency period from three days to five years. Pet. Ex. 26 at 8; Pet. Ex. 48.[15] *Tomljenovic* described ten cases of systemic lupus erythematosus ("SLE") following hepatitis B vaccine, with onset of autoimmune symptoms 56.3 days after vaccination. Pet. Ex. 26 at 8; Pet. Ex. 37.[16] *Agmon-Levin* showed onset of SLE in mice nine weeks after receipt of the hepatitis B vaccine. Pet. Ex. 26 at 8; Pet. Ex. 28.[17] *Colafrancesco* found a three-month latency period between H1N1 vaccination and the onset of Sjogren's syndrome, an autoimmune disease. Pet. Ex. 26 at 8-9; Pet. Ex. 31.[18] *Liu & Kaplowitz* discussed hypersensitivity reactions to drugs as a potential trigger for autoimmune hepatitis. Pet. Ex. 26 at 9-10; Pet. Ex. 38.[19]

Dr. Dahlgren argued that *Dodd* was the basis for the 42-day limitation and was based on a statistical study of cerebral spinal fluid changes, not clinical presentation. *Dodd* also cited *Farrington* to support the 42-day latency explaining that "[t]he point of the Farrington study is to provide a statistical model," which is a probability tool that is "complex and not easily applied to the complex interactions of vaccine triggers for autoimmune diseases like GBS." *Dodd* did report that there is a "much larger risk" of GBS following vaccination in subjects over 65, but many studies "arbitrarily used a 6-week cut off to simplify the study process . . . They could have used a longer cut-off and found additional cases." Pet. Ex. 26 at 10-11; Pet. Ex. 32[20]; Pet. Ex. 33.[21] Further, *Sevjar* discussed that the Brighton Collaboration GBS Working Group recommendation does not preclude a vaccine-induced autoimmune reaction occurring after 42 days. Pet. Ex. 26 at 11; Pet. Ex. 44.[22]

Dr. Dahlgren concluded that petitioner developed GBS from a flu vaccine, and the peer reviewed scientific literature "proves that the 42 day rule should not be applied as a hard and fast deadline." Pet. Ex. 26 at 12. The flu vaccine was a substantial contributing factor to the onset of petitioner's GBS. *Id.*

ASS'N 698 (1982), filed as "Pet. Ex. 37."

[15] Abdulla Watad et al., *The autoimmune/inflammatory syndrome induced by adjuvants (ASIA)/Shoenfeld's syndrome: descriptive analysis of 300 patients from the international ASIA syndrome registry*, 37 CLINICAL RHEUMATOLOGY 483 (2018), filed as "Pet. Ex. 48."

[16] Lucija Tomljenovic et al., *Vaccination in Autoimmune Animal Models*, ISR. MED. ASS'N J. 657 (2014), filed as "Pet. Ex. 37."

[17] Nancy Agmon-Levin et al., *Immunization with hepatitis B vaccine accelerates SLE-like disease in a murine model*, 54 J. AUTOIMMUNITY 21 (2014), filed as "Pet. Ex. 28."

[18] Serena Colafrancesco et al., *Autoimmune/Inflammatory Syndrome Induced by Adjuvants and Sjogren's Syndrome*, 18 ISR. MED. ASS'N J. 150 (2016), filed as "Pet. Ex. 31."

[19] Zhang-Xu Liu & Neil Kaplowitz, *Immune-mediated drug-induced liver disease*, 6 CLINICAL LIVER DISEASE 755 (2002), filed as "Pet. Ex. 38."

[20] Caitlin N. Dodd et al., *International collaboration to assess the risk of Guillain Barré Syndrome following Influenza A (H1N1) 2009 monovalent vaccines*, 31 VACCINE 4448 (2013), filed as "Pet. Ex. 32."

[21] C.P. Farrington, *Relative Incidence Estimation From Case Series for Vaccine Safety Evaluation*, 51 BIOMETRICS 228 (1995), filed as "Pet. Ex. 33."

[22] James J. Sejvar et al., *Guillain-Barré syndrome and Fisher syndrome: Case definitions and guidelines for collection, analysis, and presentation of immunization safety data*, 29 VACCINE 599 (2011), filed as "Pet. Ex. 44."

2. Dr. Dahlgren's Second Report

In his second report, Dr. Dahlgren again argued that 42 days is not the limit for onset of neurological symptoms of inflammatory demyelinating neuropathy following vaccine administration. Dr. Dahlgren now referred to petitioner's condition as CIDP.[23] Pet. Ex. 55 at 1-2.

Dr. Dahlgren explained that while petitioner presented to his physician reporting new onset of neurological complaints 12 weeks after vaccination, it is not clear from the records how long he had been experiencing these symptoms when he went to the doctor. Pet. Ex. 55 at 1-2. Further, Dr. Dahlgren noted that petitioner had a history of memory problems. He experienced flu-like symptoms from October 24 to 27, 2015, which he did not report to his physician in December 2015. He only reported that he had weakness for "several" days. *Id*. Therefore, the date of onset of his weakness should not be considered "as a definitive line disqualification to a conclusion that the flu vaccine was the triggering event for what was eventually diagnosed as CIDP." Pet. Ex. 55 at 2.

According to Dr. Dahlgren, GBS and CIDP have the same mechanism causing autoimmune damage to myelin, and both are treated with IVIG. The "only difference between GBS and CIDP is the duration of the illness." Pet. Ex. 55 at 2. Both have a common etiology, which is the formation of antibodies against myelin that occurs due to "a breakdown in self-recognition due to a failure in the body's immune system." *Id*. at 2-3. Individual susceptibility predisposes to this error in the immune system. Vaccines intentionally stimulate the immune system and "a rare number of individuals experience an overactive, erroneous complication of an errant immune reaction." *Id*.

Dr. Dahlgren referenced petitioner's medical records as containing diagnoses of both GBS and CIDP, indicating that his treating physicians recognized the overlap of the two diseases. He did not include citations to the medical records. Pet. Ex. 55 at 2, 3-5.

Dr. Dahlgren cited to several studies as supportive of his opinion that GBS and CIDP are the same disease, with the only difference being duration. GBS is acute and self-limited, while

[23] The petition was never amended to allege CIDP.

CIDP is ongoing and has an onset period longer than 42 days. Pet. Ex. 55 at 5-12; Pet. Ex. 56[24]; Pet. Ex. 58[25]; Pet. Ex. 59[26]; Pet. Ex. 60[27]; Pet. Ex. 63.[28]

Dr. Dahlgren concluded that petitioner developed the "autoimmune neurological condition of GBS and CIDP." Pet. Ex. 55 at 13. IVIG helped, but he had a relapse of CIDP that did not respond to therapy, and he died from complications of CIDP. His autoimmune neurological conditions were the result of the flu vaccination. *Id*.

3. Dr. Dahlgren's Third Report

In his third report, Dr. Dahlgren submitted that Dr. Jamieson failed to address the issues he raised in his first two reports, and he therefore need not respond to her opinion that GBS is not related to vaccines. Pet. Ex. 64 at 1.

Dr. Dahlgren quoted from Dr. Jamieson's report, criticizing that she did not "provide a single quote or citation about CIDP and the epidemiology showing a link of CIDP to vaccines," but rather simply suggested that GBS and CIDP are completely different diseases. Pet. Ex. 64 at 1-2. Thus, her opinion is contrary to the CDC and the vaccine injury table. *Id*.

Dr. Dahlgren further submitted that Dr. Jamieson failed to address the literature he relied on, referring only to literature that suggested there was no link between autoimmune diseases, GBS, and vaccines: "[h]er medical expertise is outdated and contrary to modern medicine theory." Pet. Ex. 64 at 2.

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[24] Stefan Blum & Pamela A. McCombe, *Genetics of Guillain-Barré syndrome (GBS) and chronic inflammatory demyelinating polyradiculopathy (CIDP): current knowledge and future directions*, 19 J. PERIPHERAL NERVOUS SYS. 88 (2014), filed as "Pet. Ex. 56." This article describes GBS as an inflammatory polyradiculoneuropathy with several subgroups and variants, while CIDP is a chronic or relapsing motor and sensory neuropathy with inflammation and demyelination of the peripheral nerves thought to be immune-mediated. Both are autoimmune diseases with genetic susceptibility.

[25] Hubertus Köller et al., *Chronic Inflammatory Demyelinating Polyneuropathy*, 352 N. ENG. J. MED. 1343 (2005), filed as "Pet. Ex. 58." This is a 2005 article discussing CIDP as characterized by symmetrical weakness in both proximal and distal muscles that progressively increases for more than two months, setting it apart from GBS, which is self-limited. CIDP can be relapsing or chronic and progressive. *Id*. at 1. The article discusses other demyelinating inflammatory neuropathies and conditions that may be concurrent with CIDP, testing, and the pathogenesis and treatments for CIDP. *Id*. at 2-3

[26] Krista Kuitwaard et al., *Recurrences, vaccinations and long-term symptoms in GBS and CIDP*¸ 14 J. PERIPHERAL NERVOUS SYS. 310 (2009), filed as "Pet. Ex. 59." This article was based on questionnaires responded to by individuals who received vaccinations and were already diagnosed with GBS or CIDP to determine if there was an increase in symptoms following the vaccination. The study noted a host of flaws.

[27] Krista Kuitwaard et al., *Letter to the Editor: Individual patients who experienced both Guillain-Barré syndrome and CIDP*, 14 J. PERIPHERAL NERVOUS SYS. 66 (2009), filed as "Pet. Ex. 60." A 2009 letter to the editor, this discussed the cases of four patients who suffered from both GBS and CIDP, concluding that this extremely uncommon occurrence in a single patient cannot be excluded and questioning whether the two diseases are part of a clinical and pathophysiological continuum instead of fully separate diseases.

[28] Paula Restrepo-Jiménez et al., *The Immunotherapy of Guillain-Barré Syndrome*, EXPERT OP. BIOLOGICAL THEORY (2018), filed as "Pet. Ex. 63." This manuscript on treatment for GBS included that GBS usually develops one to two weeks after an infection, reaching peak after two to four weeks. CIDP was not discussed.

Dr. Dahlgren argued that the Vaccine Court already determined that GBS is compensable and added it to the vaccine table as a table injury. Since the evidence supports GBS and CIDP as related, CIDP like GBS is a complication of vaccine administration.[29] Pet. Ex. 64 at 3.

Dr. Dahlgren opined, "[q]uite simply, vaccines increase the activity of the immune system, and in some rare cases, an abnormal autoimmune system attacks and damages the myelin around nerves." Pet. Ex. 64 at 3. He added that Dr. Jamieson failed to counter "that medical theory", relying on "her own naked opinion, without any foundation, except quoting herself." *Id*.

Based on his opinion that the Vaccine Court has accepted GBS as being caused by vaccinations, it is Dr. Dahlgren's opinion that the issues here are the 42-day cut off for onset and whether CIDP is a similar autoimmune disease to GBS and caused by the same mechanisms. Pet. Ex. 64 at 3. He submitted that Dr. Jamieson agreed IVIG is effective in treating both diseases, which suggests that both GBS and CIDP arise from a common mechanism. *Id*. Dr. Dahlgren concluded though inartfully stated, that "[w]hen a disease has a common mechanism and response to treatment and occurring (sic) after a common over an aberrant over stimulation of the immune system, they are related entities." *Id*. at 4.

4. Dr. Dahlgren's Fourth Report

In a fourth report, Dr. Dahlgren addressed Dr. He's opinions. He concluded that, like Dr. Jamieson, Dr. He provided nothing of substance beyond personal opinions that petitioner's illnesses were coincidence and unrelated to his vaccination. Pet. Ex. 65 at 1.

Dr. Dahlgren discussed the literature relied on by Dr. He, concluding that neither Dr. Jamieson nor Dr. He refuted the content of his three prior reports. He added that although Dr. He disagreed with his opinion that GBS and CIDP involve the same mechanism causing damage to the myelin sheath from formation of antibodies, the literature Dr. He relied on supported "the concept of autoimmunity including GBS and CIDP being caused by vaccines." Pet. Ex. 65 at 1-6. Dr. Dahlgren's opinions remained the same because the respondent's experts did not provide a scientific argument that contradicted his provided evidence. *Id*. at 6.

B. Respondent's Expert, Dr. Dara Jamieson[30]

Dr. Jamieson issued two reports. Resp. Ex. A; Resp. Ex. C.

1. Dr. Jamieson's First Report

In her first report, Dr. Jamieson detailed petitioner's medical history, including three days of severe flu symptoms approximately one month after his receipt of the flu vaccine. Resp. Ex. A

[29] The Court's decisions about a vaccine and a particular condition or injury have no bearing on if or when a vaccine is added to a Table or whether an injury is determined to be a Table injury. *See* § 300aa-14(c).

[30] Dr. Jamieson is a board-certified neurologist. Resp. Ex. B. She received her medical degree from the University of Pennsylvania and completed her residency in neurology at the University of Pennsylvania Hospital, as well as a fellowship at the Cerebrovascular Research Center in the University of Pennsylvania School of Medicine's Department of Neurology. *Id*. She is currently a Clinical Associate Professor of Neurology at Weill Cornell Medicine. *Id*.

at 2-10. She noted the onset of petitioner's neurological symptoms as between 12 and 13 weeks after his vaccination with worsening extended over more than the next two months. *Id*. at 11-12. Petitioner had no "obvious motor or sensory deficits" when he was examined on December 31, 2016. Weakness was first noted on examination on January 11, 2017, and became more pronounced thereafter. *Id*. at 12.

In Dr. Jamieson's opinion, petitioner was misdiagnosed with cervical myelopathy and underwent an unnecessary cervical decompression surgery. Resp. Ex. A at 12. He was then treated with steroids for motor and sensory deficits which are beneficial in treating CIDP, but not GBS. When the steroids were discontinued, his weakness worsened and IVIG was initiated, which improved his extremity weakness. Petitioner suffered a remitting and relapsing course that is indicative of CIDP and required regular IVIG treatments. *Id*. Unfortunately, other intervening and unrelated medical conditions including dementia, atrial fibrillation, deep vein thrombosis, congestive heart failure, severe coronary artery disease, severe mitral valve regurgitation, and hypertension worsened his general health. *Id*.

Dr. Jamieson opined that petitioner's illness course was the classic pattern of CIDP, with relapsing and remitting symptoms over a three-year period. Resp. Ex. A at 12. Petitioner's course was distinguishable from GBS, which reaches nadir within four weeks of onset, while petitioner experienced a slow initial neurological deterioration over more than two months. In her opinion, petitioner never had GBS. *Id*. at 12-13.

Dr. Jamieson explained that the first symptoms of GBS are numbness, paresthesia, weakness, pain in the limbs, or a combination of these. The main feature is the bilateral and relatively symmetric weakness of the limbs that progresses over a period of 12 hours to 28 days before a plateau is reached. Resp. Ex. A at 13; Resp. Ex. A, Tab 3.[31] It is believed that the demyelination of the peripheral nerves in GBS/AIDP is related to a preceding triggering event, most commonly an infection but also surgery, trauma, vaccination, or bone marrow transplantation, and the immune response to this event is directed toward the myelin or axon of the peripheral nerve, resulting in demyelinating, axonal, or mixed forms of GBS. Resp. Ex. A at 13. GBS is often post-infectious with onset of rapidly progressive and monophasic illness shortly after illness, with two thirds of patients reporting preceding respiratory or gastrointestinal symptoms within four weeks of onset of weakness and usually ten to 14 days after infection. *Id*. Limb weakness, which may include sensory and/or cranial nerve symptoms, develops a week or two after immune stimulation and progresses to clinical deficit within two to four weeks. IVIG or plasma exchange ("PLEX") are used as treatment for GBS/AIDP, and unlike CIDP, these conditions do not respond to steroids. Most GBS patients make a full recovery, and reoccurrence is exceedingly rare. *Id*. at 13-14.

Dr. Jamieson pointed out that CIDP is distinctively different from GBS. CIDP is an immune-mediated peripheral neuropathy that generally presents with gradual worsening, symmetrical numbness and tingling followed by hand, feet, upper arm, and thigh motor weakness. Extremity pain and abnormal sensation can occur. There is also obvious motor deficit, less prominent than the sensory loss, and decreased or absent reflexes. Resp. Ex. A at 14.

[31] Nobuhiro Yuki & Hans-Peter Hartung, *Guillain-Barré Syndrome*, 366 N. ENG. J. MED. 2294 (2012), filed as "Resp. Ex. A, Tab 3."

Unlike the rapidly evolving GBS, CIDP develops slowly over more than eight weeks, and the evolution of initial symptoms can take at least two months to reach clinical nadir from onset. Resp. Ex. A at 14. Deficits involving respiratory muscles, autonomic function, and cranial nerve are less prominent in CIDP. CIDP also generally does not have an immunological trigger, like a prior infection in GBS. *Id*. at 15.

Dr. Jamieson agreed that GBS and CIDP have immune pathophysiology with similar findings on EMG/NCV studies depending on the timing of the studies, but the specific antigenic targets, immune effectors and responses to treatment are different. Resp. Ex. A at 15. The pathogenesis of CIDP is poorly understood but appears to involve both humoral immunity[32] and cellular immunity[33] given its chronic course, while the rapid course of GBS suggests only humoral immunity is involved. These are "distinctly different diseases," so that the pathophysiological and epidemiological attributes of GBS and CIDP cannot be extrapolated to the other. *Id*. at 15-16.

Finally, CIDP is treating with IVIG with steroids and PLEX, while steroids are not used to treat GBS. Both respond to IVIG and PLEX, though clinical improvement is permanent for GBS but only transient for CIDP. Chronic steroid or steroid sparing medications are not used to treat GBS because of the lack of response and other treatment options, but these are used to treat CIDP. Resp. Ex. A at 16.

Dr. Jamieson summarized that arguments about an association between GBS and influenza vaccine are not applicable to CIDP because they are different neurological diseases. Resp. Ex. A at 16. Beyond the 1976 H1N1 vaccination program, "there has been no consistently proven association" between flu vaccine and the development of GBS. *Id*. Dr. Jamieson detailed the many studies regarding GBS and flu vaccine noting that only case reports contained instances of CIDP following vaccination. *Id*. at 16-17. Further, the CDC found the evidence inadequate to accept or reject a causal relationship between the flu vaccine and CIDP. *Id*. at 18-19.

Dr. Jamieson concluded that petitioner developed CIDP, not GBS, that developed three months after the flu vaccine. GBS and CIDP are two distinct peripheral neuropathies, and conflating their immunological similarities and vaccination associations is not supported by the evidence. Resp. Ex. A at 20. There is no immunological or epidemiological evidence that a temporally remote flu vaccine can trigger CIDP, a disease not identified as caused or triggered by vaccinations. It would be more likely that his flu-like syndrome two months before was the trigger or contributing factor, if there was one. *Id*. at 20-21.

2. Dr. Jamieson's Second Report

Dr. Jamieson responded to Dr. Dahlgren in her second report. Resp. Ex. C. She noted that the literature Dr. Dahlgren relied on distinguishes GBS and CIDP, as stated in her initial report.

[32] Humoral immunity is immunity mediated by antibodies. *Humoral immunity*, DORLAND'S 906.

[33] Cellular or cell-mediated immunity is "immunity mediated by T lymphocytes either through release of lymphokines or through exertion of direct cytotoxicity, transmissible by transfer of lymphocytes but not serum; it includes type IV hypersensitivity reactions and systemic responses to viral or microbial infections or to tumor cells." *Cell-mediated immunity*, DORLAND'S 906.

Resp. Ex. C at 1-2; Pet. Ex. 56[34]; Pet. Ex. 58.[35] Further, she argued that Dr. Dahlgren's opinion that the fact that GBS and CIDP can both be treated with IVIG suggests a common mechanism is like suggesting that asthma and MS have a common mechanism because both are treated with steroids: "Treatment of two diseases with a commonly used immunotherapy does not imply an overlapping pathophysiological mechanism." *Id*. at 2. Many unrelated autoimmune diseases are treated with the same therapies. Arguably, if the response to treatment defined pathophysiological mechanism, then the difference in response to steroid between GBS and CIDP mitigates their commonality. *Id*.

Dr. Jamieson wrote that Dr. Dahlgren "criticizes the use of epidemiological evidence to refute an association between GBS and vaccination, yet he sends only references of a few case reports of supposed CIDP after vaccination to support a linkage." Not only were his references unpersuasive of his opinion, but some were also referenced in her report in support of her opinions. Resp. Ex. C at 2-3; Pet. Ex. 57[36]; Pet. Ex. 59[37]; Pet. Ex. 62.[38]

Dr. Jamieson agreed that the issues in the case are the onset far in excess of 42 days and whether CIDP and GBS are similar autoimmune diseases caused by the same mechanism, and she maintained that GBS and CIDP are distinct autoimmune disease that cannot be conflated. Further, she stated that Dr. Dahlgren failed to provide any literature that supports a causative link between an influenza vaccination and CIDP developing three months later, or that a distinct disease with a supposed vaccine association within a certain timeframe can be replaced by a different disease outside that timeframe. Resp. Ex. C at 3.

Dr. Jamieson reiterated that there is no support for an association between a flu vaccine and onset of CIDP symptoms three months later, and that petitioner's flu-like symptoms two months prior to the onset were more likely the cause of his chronic neuropathy. Resp. Ex. C at 3.

C. Respondent's Expert, Dr. You-Wen He[39]

Dr. He issued one report. Resp. Ex. D.

Dr. He detailed petitioner's medical history, specifically his receipt of a high dose flu vaccine on September 27, 2016, his severe flu-like symptoms one month later that were treated with over-the-counter medications, and the development of symptoms of GBS and CIDP three months after receipt of the flu vaccination. He further noted that petitioner had a family history of

[34] Blum & McCombe, *supra* note 24.
[35] Köller et al., *supra* note 25.
[36] J.M. Brostoff et al., *Post-influenza vaccine chronic inflammatory demyelinating polyneuropathy*, 37 AGE & AGING 229 (2007), filed as "Pet. Ex. 57."
[37] Kuitwaard et al., *supra* note 26.
[38] Gauthier Remiche et al., *Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) associated to hereditary neuropathy with liability to pressure palsies (HNPP) and revealed after influenza AH1N1 vaccination*, 113 ACTA NEUROLOGICA BELGICA 519 (2013), filed as "Pet. Ex. 63."
[39] Dr. He received his medical degree in China from the Fourth Military Medical University and his Ph.D. in microbiology and immunology from the University of Miami. Resp. Ex. E. He has been a professor of immunology in the Duke University Medical Center Department of Immunology since 2000. *Id*.

autoimmune diseases including a sister with lupus and a grandmother with diabetes, thyroid disease, arthritis, and lupus. Resp. Ex. D at 3; Pet. Ex 4 at 3.

Dr. He described GBS as an inflammatory polyneuropathy characterized by acute onset, rapid progression, symmetrical weakness, and hyporeflexia or areflexia. Resp. Ex. D at 3. GBS is post-infectious, with two thirds of patients reporting either respiratory or gastrointestinal tract infection before onset. *Id*. at 4; Resp. Ex. D, Tab 1[40]; Resp. Ex. D, Tab 3.[41] GBS has been associated with campylobacter jejuni, cytomegalovirus, Epstein-Barr virus, mycoplasma pneumonia, haemophilus influenza, influenza A virus, enterovirus, hepatitis E, varicella zoster, and Zika virus. Resp. Ex. D at 4; Resp. Ex. D, Tab 1[42]; Resp. Ex. D, Tab 3[43]; Resp. Ex. D, Tab 5.[44] Although not firmly established, cross-reactive autoantibodies may play a role in the immunopathogenesis of GBS, attacking peripheral nerves and causing demyelination and axonal damage. Resp. Ex. D at 4; Resp. Ex. D, Tab 1.[45]

Dr. He described CIDP as an acquired, immune-mediated neuropathy affecting peripheral nerves and nerve roots, characterized by a relapsing and remitting or progressive course that is steroid responsive and has electrodiagnostic or pathologic features of demyelination. The cause and specific immunologic triggers are unclear. Resp. Ex. D at 5; Resp. Ex. D, Tab 9.[46] Evidence beyond a temporal relationship between vaccination and the development of CIDP has not been provided in any literature. Resp. Ex. D at 5; Resp. Ex. D, Tab 8.[47]

Dr. He opined that while it is generally accepted that H1N1 influenza vaccines have been associated with an increased risk GBS, other seasonal influenza vaccines have not. Resp. Ex. D at 4-5. Dr. He then addressed Dr. Dahlgren's opinions and the papers he relied upon. Resp. Ex. D at 6-8; Pet. Ex. 30[48]; Pet. Ex. 34[49]; Pet. Ex. 35[50]; Pet. Ex. 36[51]; Pet. Ex. 39[52]; Pet. Ex. 41[53]; Pet. Ex.

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[40] Bianca van den Berg et al., *Guillain-Barré syndrome: pathogenesis, diagnosis, treatment and prognosis*, 10 NAT'L REV. NEUROLOGY 469 (2014), filed as "Resp. Ex. D, Tab 1."
[41] Peter D. Donofrio, *Guillain-Barré Syndrome*, 23 CONTINUUM 1295 (2017), filed as "Resp. Ex. D, Tab 3."
[42] van den Berg et al., *supra* note 40.
[43] Donofrio, *supra* note 41.
[44] Badrul Islam et al., *Guillain-Barré syndrome following varicella-zoster virus infection*, 37 EUR. J. CLINICAL MICROBIOLOGY & INFECTIOUS DISEASES 511 (2018), filed as "Resp. Ex. D, Tab 5."
[45] van den Berg et al., *supra* note 40.
[46] Richard A. Lewis & Suraj Ashok Muley, *Chronic inflammatory demyelinating polyneuropathy: Etiology, clinical features, and diagnosis*, UPTODATE (Nov. 23, 2021, 1:57 PM), https://www.uptodate.com/contents/chronic-inflammatory-demyelinating-polyneuropathy-etiology-clinical-features-and-diagnosis, filed as "Resp. Ex. D, Tab 9."
[47] INST. OF MED., ADVERSE EFFECTS OF VACCINES: EVIDENCE & CAUSALITY (Kathleen Stratton et al. eds., 2012), filed as "Resp. Ex. D, Tab 8."
[48] L.H. Martín Arias et al., *Guillain-Barré syndrome and influenza vaccines: A meta-analysis*, 33 VACCINE 3773 (2015), filed as "Pet. Ex. 30."
[49] Penina Haber et al., *Guillain-Barré Syndrome Following Influenza Vaccination*, 292 J. AM. MED. ASS'N 2478 (2004), filed as "Pet. Ex. 34."
[50] Penina Haber et al., *Post-licensure surveillance of quadrivalent inactivated influenza (IIV4) vaccine in the United States, Vaccine Adverse Event Reporting System (VAERS), July 1,2013—May 31, 2015*, 34 VACCINE 2507 (2016), filed as "Pet. Ex. 35."
[51] Eugene S. Hurwitz et al., *Guillain-Barré Syndrome and the 1978-1979 Influenza Vaccine*, 304 N.E. J. MED. 1557 (1981), filed as "Pet. Ex. 36."
[52] Marks & Halpin, *supra* note 12.
[53] Daniel A. Salmon et al., *Association between Guillain-Barré syndrome and influenza A (H1N1) 2009 monovalent inactivated vaccines in the USA: a meta-analysis*, 381 LANCET 1461 (2013), filed as "Pet. Ex. 41."

42.[54] Dr. He summarized that two of the papers, *Marks & Halpin* and *Salmon*, reported the increased risk of GBS following receipt of the pandemic swine flu vaccine. The other five studies did not support a causal relationship between the seasonal flu vaccine and GBS, and there were no reports filed of GBS caused by Fluzone, the vaccine petitioner received. Resp. Ex. D at 8.

Further, Dr. He asserted that Dr. Dahlgren ignored two important potential contributing factors for petitioner's alleged GBS and CIDP: the severe flu symptoms he reported as occurring on October 24, 2016, and his family history of autoimmune disease making petitioner genetically predisposed to develop autoimmune diseases such as GBS and CIDP. He added that it was "extremely unlikely" that an immune response to a flu vaccine could result in onset of GBS and CIDP three months later. Resp. Ex. D at 8.

Dr. He explained that latency period is defined as the "the time that passes between being exposed to something that can cause disease (such as radiation or a virus) and having symptoms." Resp. Ex. D at 9. He argued that Dr. Dahlgren incorrectly used this term, and that calling a time period for a disease to develop "latency" indicates an established causal relationship, which there is not between the seasonal flu vaccine and GBS and CIDP. *Id*.

Dr. He stated that the primary immune response to an antigen reaches peak level at ten to 17 days and a secondary response to the same antigen reaches peak level in two to seven days after priming, then starts to decline immediately thereafter. Resp. Ex. D at 9. With flu vaccination, antibody-secreting cells peak at seven to eight days then rapidly decline, with low numbers of antibody-secreting cells detected at 11 days post-vaccination. In less than three weeks, antigen-specific T cells undergo a contraction phase, forming a stable memory T cell pool. Resp. Ex. D at 9; Resp. Ex. D, Tab 10[55]; Resp. Ex. D, Tab 11.[56] Summarily, an immune response to a foreign antigen completes its process in about three weeks, and a six-week timeframe covers two cycles of the immune response. Therefore, six weeks or 42 days is "undoubtedly more than enough time" for a vaccine-induced immune response to occur. Resp. Ex. D at 9. The 1976 swine flu vaccination campaign showed no increased risk of GBS beyond six weeks, consistent with this time frame. Therefore, it is unlikely that the flu vaccine triggered the onset of petitioner's alleged GBS and subsequent CIDP symptoms three months later. *Id*; Resp. Ex. D, Tab 12.[57]

Dr. He addressed the literature relied on by Dr. Dahlgren in detail. Resp. Ex. D at 10. He noted that *Prestel*[58] investigated the Miller Fisher variant of GBS after the 2009 H1N1 pandemic vaccination, not a seasonal flu vaccine, and these are very different because the seasonal flu vaccine does not contain adjuvants. Further, the risk period was defined as five to 42 days after vaccination while the control period was 43-150 days, and cases that occurred at days 43-150 were

[54] Sukhminder K. Sandhu et al., *Near real-time surveillance for Guillain-Barré syndrome after influenza vaccination among the Medicare population, 2010/11 to 2013/14*, 35 VACCINE 2986 (2017), filed as "Pet. Ex. 42."
[55] Rebecca J. Cox et al., *An early humoral immune response in peripheral blood following parenteral inactivated influenza vaccination*, 12 VACCINE 993 (1994), filed as "Resp. Ex. D, Tab 10."
[56] Matthew A. Williams & Michael J. Bevan, *Effector and Memory CTL Differentiation*, 25 ANN. REV. IMMUNOLOGY 171 (2007), filed as "Resp. Ex. D, Tab 11."
[57] Thomas J. Safranek et al., *Reassessment of the Association between Guillain-Barré Syndrome and Receipt of Swine Influenza Vaccine in 1976-1977: Results of a Two-State Study*, 133 AM. J. EPIDEMIOLOGY 940 (1991), filed as "Resp. Ex. D, Tab 12."
[58] Prestel et al., *supra* note 10.

studied as vaccination-unrelated controls, with no increased risk of GBS following the swine flu vaccine found beyond six weeks. Dr. He also noted that Dr. Dahlgren misinterpreted Table 4 of *Prestel*, explaining that age was not a modifier and there was no difference between age groups in terms of GBS risk following Pandemix vaccination. *Id*. at 10-11. Similarly, *Kaplan*[59] suggested no increased risk of GBS with flu vaccines, noting that the "trigger agent" present in the 1976 swine flu vaccine was not present in subsequent flu vaccines. *Id*. at 12. *Marks & Halpin*[60] discussed the 1976 swine flu pandemic having an increased risk of GBS within six weeks of swine flu vaccination but no increased risk beyond 6 weeks, and *Schonberger*[61] reported GBS after the 1976 swine flu vaccine with an increased risk concentrated primarily within the five-week period after vaccination. *Id*. at 11-12.

Watad[62] had no relevance to flu vaccine and GBS and studied the relationship between ASIA and adjuvants, but petitioner's flu vaccine did not have an adjuvant. Resp. Ex. D at 13. *Soriano*[63], *Tomljenovic*[64], *Agmon-Levin*[65], and *Liu & Kaplowitz*[66] all studied vaccines other than the flu vaccine and diseases other than GBS. *Id*. at 11, 13-15. According to Dr. He, Dr. Dahlgren mischaracterized *Colafranceso*[67], which did not study any disease latency as he alleged. Resp. Ex. D at 14.

Dr. He detailed the differences between an immune response to a viral infection versus that to a vaccination, concluding that the fact that petitioner experienced severe flu symptoms a month after his vaccination suggests that the infection, not the vaccination, "is a much more likely trigger of his alleged GBS and CIDP." Resp. Ex. D at 15-16. Dr. He claimed that Dr. Dahlgren failed to discuss this illness and misinterpreted the studies he relied on to argue that a three-month onset following vaccination was reasonable. *Id*. at 19.

Dr. He then addressed Dr. Dahlgren's supplemental report and opinions related to the flu vaccine and CIDP. He noted that *Köller*[68] summarized different aspects of CIDP but did not discuss any information related to vaccination, and *Restrepo-Jimenez*[69] discussed immunotherapy for GBS, not vaccines. Resp. Ex. D at 19. According to Dr. He, updated literature indicates that in the majority of patients, the specific immunologic triggers or causes of CIDP remain unclear, and no specific predisposing risk factors for CIDP have been identified. *Blum & McCombe*[70] did not present any evidence to suggest any relationship between vaccination and CIDP. Resp. Ex. D at 20; Resp. Ex. D, Tab 9.[71] Dr. He agreed that there need to be more studies comparing GBS and CIDP in order to understand the pathogenesis of the two diseases. Resp. Ex. D at 20.

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[59] Kaplan et al., *supra* note 14.
[60] Marks & Halpin, *supra* note 12.
[61] Schonberger et al., *supra* note 13.
[62] Watad et al., *supra* note 15.
[63] Soriano et al., *supra* note 11.
[64] Tomljenovic et al., *supra* note 16.
[65] Agmon-Levin et al., *supra* note 17.
[66] Liu & Kaplowitz et al., *supra* note 19.
[67] Colafrancesco et al., *supra* note 18.
[68] Köller et al., *supra* note 25.
[69] Restrepo-Jiménez et al., *supra* note 28.
[70] Blum & McCombe, *supra* note 24.
[71] Lewis & Muley, *supra* note 46.

Dr. He submitted that the *Kuitwaard*[72] study did not investigate vaccinations as a cause of CIDP or GBS, but rather whether vaccinations led to recurrences of GBS or increased disability in CIDP in patients who were already diagnosed with GBS or CIDP. The study found no recurrence or increased symptoms in GBS and increased symptoms in a fraction of CIDP patients, with no evidence to suggest a causal link. Resp. Ex. D at 21. Further, the study supported genetics as a factor in autoimmune disease, which supports that petitioner's family history of autoimmune disease was a more important factor in his development of GBS and CIDP than the vaccine. *Id*.

Dr. He opined that the second *Kuitwaard*[73] study did not support Dr. Dahlgren's claim that there is an overlap between GBS and CIDP. Resp. Ex. D at 21. One of the four patients that the article focused on developed GBS following a flu vaccination at age 39 and developed CIDP at age 68. He added that it was unclear which flu vaccine the patient received at age 39 and what his history was in the 29 years between the onset of GBS and CIDP, and it was just a reporting of temporal relation. *Id.* at 21-22. Dr. He stated that the IOM concluded that *Brostoff*[74] and *Pritchard*[75] did not provide any mechanistic evidence beyond a temporal relationship and therefore cannot be used as evidence to draw causal conclusions. *Id*. at 22.

Dr. He summarized that Dr. Dahlgren cited references showing results of disease recurrences after flu vaccination and a case report of CIDP after H1N1 vaccination, but "did not provide convincing evidence" to connect seasonal flu vaccines including Fluzone to GBS or CIDP after three months. Resp. Ex. D at 23. He concluded that Dr. Dahlgren did not provide any reliable evidence to support his theory that the Fluzone vaccine caused petitioner's GBS and/or CIDP that led to his death. *Id*. at 24.

V. Standards for Adjudication

A. Legal Standard Regarding Causation

The Vaccine Act provides two avenues for petitioners to receive compensation. First, a petitioner may demonstrate a "Table" injury—i.e., an injury listed on the Vaccine Injury Table that occurred within the provided time period. 42 U.S.C. § 300aa-11(c)(1)(C)(i). "In such a case, causation is presumed." *Capizzano v. Sec'y of Health & Human Servs*., 440 F.3d 1317, 1320 (Fed. Cir. 2006); *see* § 13(a)(1)(B). Second, where the alleged injury is not listed on the Vaccine Injury Table, a petitioner may demonstrate an "off-Table" injury, which requires that the petitioner "prove by a preponderance of the evidence that the vaccine at issue caused the injury." *Capizzano*, 440 F.3d at 1320; *see* § 11(c)(1)(C)(ii); *see also Wright v. Sec'y of Health & Human Servs*., 22 F.4th 999, 1006 (Fed. Cir. 2022) (defining the term "residual effects" in the Act, as "detrimental conditions within the patient, such as lingering or recurring signs and symptoms" of the alleged vaccine injury, which are compensable). A petitioner need not show that the vaccination was the sole cause, or even the predominant cause, of the alleged injury; showing that the vaccination was

[72] Kuitwaard et al., *supra* note 26.
[73] Kuitwaard et al., *supra* note 27.
[74] Brostoff et al., *supra* note 35.
[75] J. Pritchard et al., *Risk of relapse of Guillain-Barré syndrome or chronic inflammatory demyelinating polyradiculoneuropathy following immunization*, 73 J. NEUROLOGY, NEUROSURGERY, & PSYCHIATRY 348 (2002), filed as "Pet. Ex. 61."

a "substantial factor" and a "but for" cause of the injury is sufficient for recovery. *Pafford v. Sec'y of Health & Human Servs*., 451 F.3d 1352, 1355 (Fed. Cir. 2006); *Shyface v. Sec'y of Health & Human Servs*., 165 F.3d 1344, 1352 (Fed. Cir. 1999). Petitioners are not required "to eliminate alternative causes as part of establishing [their] prima facie case." *Doe v. Sec'y of Health & Human Servs*., 601 F.3d 1349, 1357-58 (Fed. Cir. 2010); *see Walther v. Sec'y of Health & Human Servs*., 485 F.3d 1146, 1152 (Fed. Cir. 2007) (holding that a "petitioner does not bear the burden of eliminating alternative independent potential causes"). Once a petitioner has proven causation by preponderant evidence, "the burden then shifts to the respondent to show by a preponderance of the evidence that the injury is due to factors unrelated to the administration of the vaccine." *Deribeaux ex rel. Deribeaux v. Sec'y of Health & Human Servs*., 717 F.3d 1363, 1367 (Fed. Cir. 2013) (citing 42 U.S.C. § 300aa-13(a)(1)(B)).

To prove causation, a petitioner must satisfy the three-pronged test established in *Althen v. Sec'y of Health & Human Servs*., 418 F.3d 1274 (Fed. Cir. 2005). *Althen* requires that a petitioner show by preponderant evidence that a vaccination they received caused their injury "by providing: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." *Id*. at 1278. Together, these prongs must show "that the vaccine was 'not only a but-for cause of the injury but also a substantial factor in bringing about the injury.'" *Stone v. Sec'y of Health & Human Servs*., 676 F.3d 1373, 1379 (Fed. Cir. 2012) (quoting *Shyface*, 165 F.3d at 1352-53). Causation is determined on a case-by-case basis, with "no hard and fast per se scientific or medical rules." *Knudsen v. Sec'y of Health & Human Servs*., 35 F.3d 543, 548 (Fed. Cir. 1994). Petitioners are not required to identify "specific biological mechanisms" to establish causation, nor are they required to present "epidemiologic studies, rechallenge, the presence of pathological markers or genetic disposition, or general acceptance in the scientific or medical communities." *Capizzano*, 440 F.3d at 1325 (quoting *Althen*, 418 F.3d at 1280). "[C]lose calls regarding causation are resolved in favor of injured claimants." *Althen*, 418 F.3d at 1280.

Each *Althen* prong requires a different showing. Under the first prong, a petitioner must provide a "reputable medical theory" demonstrating that the vaccine received can cause the type of injury alleged. *Pafford*, 451 F.3d at 1355-56 (citation omitted). To satisfy this prong, a petitioner's "theory of causation must be supported by a 'reputable medical or scientific explanation.'" *Andreu*, 569 F.3d at 1379 (quoting *Althen*, 418 F.3d at 1278). This theory need only be "legally probable, not medically or scientifically certain." *Id*. at 1380 (emphasis omitted) (quoting *Knudsen*, 35 F.3d at 548). Nevertheless, "petitioners [must] proffer trustworthy testimony from experts who can find support for their theories in medical literature." *LaLonde*, 746 F.3d at 1341.

The second *Althen* prong requires proof of a "logical sequence of cause and effect." *Capizzano*, 440 F.3d at 1326 (quoting *Althen*, 418 F.3d at 1278). Even if the vaccination can cause the injury, a petitioner must show "that it did so in [this] particular case." *Hodges v. Sec'y of Health & Human Servs*., 9 F.3d 958, 962 n.4 (Fed. Cir. 1993) (citation omitted). "A reputable medical or scientific explanation must support this logical sequence of cause and effect," *Id*. at 961 (citation omitted), and "treating physicians are likely to be in the best position to determine whether a logical sequence of cause and effect show[s] that the vaccination was the reason for the injury,"

Paluck v. Sec'y of Health & Human Servs., 786 F.3d 1373, 1385 (Fed. Cir. 2015) (quoting *Andreu*, 569 F.3d at 1375).

The third *Althen* prong requires that a petitioner establish a "proximate temporal relationship" between the vaccination and the alleged injury. *Althen*, 418 F.3d at 1281. This "requires preponderant proof that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." *De Bazan v. Sec'y of Health & Human Servs*., 539 F.3d 1347, 1352 (Fed. Cir. 2008). Typically, "a petitioner's failure to satisfy the proximate temporal relationship prong is due to the fact that onset was too late after the administration of a vaccine for the vaccine to be the cause." *Id*. However, "cases in which onset is too soon" also fail this prong; "in either case, the temporal relationship is not such that it is medically acceptable to conclude that the vaccination and the injury are causally linked." *Id*.; *see also Locane v. Sec'y of Health & Human Servs*., 685 F.3d 1375, 1381 (Fed. Cir. 2012) ("[If] the illness was present before the vaccine was administered, logically, the vaccine could not have caused the illness.").

B. Legal Standard Regarding Fact Finding

The process for making factual determinations in Vaccine Program cases begins with analyzing the medical records, which are required to be filed with the petition. § 11(c)(2). Medical records created contemporaneously with the events they describe are presumed to be accurate and "complete" such that they present all relevant information on a patient's health problems. *Cucuras v. Sec'y of Health & Human Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993). In making contemporaneous reports, "accuracy has an extra premium" given that the "proper treatment hang[s] in the balance." *Id.* Contemporaneous medical records that are clear, consistent, and complete warrant substantial weight "as trustworthy evidence." *Id*. Indeed, "where later testimony conflicts with earlier contemporaneous documents, courts generally give the contemporaneous documentation more weight." *Campbell ex rel. Campbell v. Sec'y of Health & Human Servs.*, 69 Fed. Cl. 775, 779 (2006); *see United States v. U.S. Gypsum Co.*, 333 U.S. 364, 396 (1948). But petitioners can support their claim with oral testimony if it is credible and consistent with the medical records. *See, e.g.*, *Stevenson ex rel. Stevenson v. Sec'y of Health & Human Servs.*, No. 90-2127V, 1994 WL 808592, at *7 (Fed. Cl. Spec. Mstr. June 27, 1994) (crediting the testimony of a fact witness whose "memory was sound" and "recollections were consistent with the other factual evidence"). In short, "the record as a whole" must be considered. § 13(a).

C. Evaluating Expert Testimony

Establishing a sound and reliable medical theory connecting the vaccine to the injury often requires a petitioner to present expert testimony in support of his or her claim. *Lampe v. Sec'y of Health & Human Servs.*, 219 F.3d 1357, 1361 (Fed. Cir. 2000). The Supreme Court's opinion in *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993), requires that courts determine the reliability of an expert opinion before it may be considered as evidence. "In short, the requirement that an expert's testimony pertain to 'scientific knowledge' establishes a standard of evidentiary reliability." *Id*. at 590 (citation omitted). Thus, for Vaccine Act claims, a "special master is entitled to require some indicia of reliability to support the assertion of the expert witness." *Moberly ex rel. Moberly v. Sec'y of Health & Human Servs.*, 592 F.3d 1315, 1324 (Fed.

Cir. 2010). The *Daubert* factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Human Servs.*, 94 Fed. Cl. 53, 66-67 (2010) ("uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted"). Where both sides offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen*, 618 F.3d at 1347 (citing *Lampe*, 219 F.3d at 1362). And nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder ex rel. Snyder v. Sec'y of Health & Human Servs.*, 88 Fed. Cl. 706, 743 (2009) (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997)).

D. Consideration of Medical Literature

Finally, although this decision discusses some but not all the literature in detail, the undersigned reviewed and considered all of the medical records and literature submitted in this matter. *See Moriarty ex rel. Moriarty v. Sec'y of Health & Human Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("We generally presume that a special master considered the relevant record evidence even though [s]he does not explicitly reference such evidence in h[er] decision."); *Simanski v. Sec'y of Health & Human Servs.*, 115 Fed. Cl. 407, 436 (2014) ("[A] Special Master is 'not required to discuss every piece of evidence or testimony in her decision.'" (citation omitted)), *aff'd*, 601 F. App'x 982 (Fed. Cir. 2015).

VI. Discussion

A. Petitioner's Diagnosis

As a threshold matter, petitioner must first establish that he actually suffered the injury alleged in the petition. *See Broekelschen v. Sec'y of Health and Human Servs.*, 618 F.3d 1339, 1346 (Fed. Cir. 2010). As the Federal Circuit precedent establishes, in certain cases it is appropriate to determine the nature of the petitioner's injury before engaging in the *Althen* analysis. *Id*. Thus, where "the existence and nature of the injury itself is in dispute, it is the special master's duty to *first determine* which injury is best supported" by the evidence. *Lombardi v. Sec'y of Health and Human Servs.*, 656 F.3d 1343, 1352 (Fed. Cir. 2011) (affirming a special master's decision to dismiss a petition when the petitioner could not establish that she had any of the three diagnoses alleged) (citing *Broekelschen*, 618 F.3d at 1345) (emphasis added).

1. Overview of GBS and CIDP

Guillain-Barre Syndrome ("GBS") is an acute autoimmune disorder of the peripheral nerves characterized primarily by muscle weakness and loss of reflexes. Pet. Ex. 30 at 1.[76] GBS is often associated with a preceding gastrointestinal or upper respiratory tract infection, including influenza, generally 10-14 days prior to onset. About 70% of patients report preceding illness, though it is often benign and may be minimized or forgotten by the patient. *Id*.; Resp. Ex. A, Tab

[76] Martín Arias et al., *supra* note 48.

4 at 1.[77] The exact causes of GBS are unknown, but it is thought to be triggered by antigenic stimulation resulting in demyelination and damage to the peripheral nerves. Pet. Ex. 30 at 1.[78] GBS has several subgroups that are described by corresponding diagnostic criteria and can be divided on pathological grounds into acute inflammatory demyelinating polyradiculoneuropathy ("AIDP") and acute motor axonal neuropathy ("AMAN"). There are also variants of GBS such as Miller Fisher Syndrome Pet. Ex. 56 at 1.[79] GBS evolves over days, often beginning with numbness and weakness in the lower limbs. The weakness can be rapid, resulting in quadriplegia within days. Approximately 50% of GBS patients experience maximum weakness by two weeks, 80% by three weeks, and 90% by four weeks. Progression beyond four weeks is unusual and raises concern for other illnesses, particularly CIDP. Resp. Ex. A, Tab 4 at 2.[80] Essentially all patients will have areflexia or at least hyporeflexia at some point during the illness. About 50% will develop facial weakness or other cranial nerve issues, and 30% develop respiratory failure and require intubation and ventilation. *Id*. The Brighton Collaboration Diagnostic Criteria for GBS includes: bilateral and flaccid weakness of limbs; decreased or absent deep tendon reflexes in the weak limbs; monophasic illness and interval between onset and nadir of weakness between 12 hours and 28 days and subsequent clinical plateau; electrophysiologic findings consistent with GBS; albuminocytologic dissociation – elevation of the CSF protein level above laboratory normal value and CSF total white blood cell count less than 50 cells/ 2µL; and absence of an identified alternative diagnosis for weakness. *Id*. at 6. Features that cast doubt or eliminate GBS as the diagnosis include marked persistent asymmetry of weakness, bowel or bladder dysfunction at onset, the presence of greater than 50 cells/ 2µL in CSF, sharp sensory level, severe pulmonary dysfunction with little or no limb weakness at onset, fever at onset, and/or slow progression of weakness more than four weeks. *Id*. at 7.

Chronic inflammatory demyelinating polyneuropathy ("CIDP") is a chronic or relapsing motor and sensory neuropathy, with inflammation and demyelination of the peripheral nerve. Pet. Ex. 56 at 1.[81] It is characterized by symmetrical weakness in both proximal and distal muscles that progressively increases for more than two months, setting it apart from GBS, which is self-limited. Pet. Ex. 58 at 1.[82] CIDP is associated with impaired sensation, absent or diminished tendon reflexes, an elevated cerebrospinal fluid protein level, demyelinating nerve conduction studies, and signs of demyelination in nerve biopsy specimens. The course can be relapsing or chronic and progressive. *Id*. CIDP responds well to corticosteroid treatment, distinguishing it from other forms of acquired demyelinating polyneuropathies. *Id*. at 1-2. The cause of CIDP is unknown, although abnormalities in both cellular and humoral immunity have been shown. Pet. Ex. 70 at 1.[83]

Both GBS and CIDP are considered immune-mediated polyneuropathies associated with a variable clinical courses and outcomes. There have been reports of rare patients affected by both GBS and CIDP, suggesting that GBS and CIDP may constitute a continuum or that there are

[77] Donofrio, *supra* note 41.
[78] Martín Arias et al., *supra* note 48.
[79] Blum & McCombe, *supra* note 24.
[80] Donofrio, *supra* note 41.
[81] Blum & McCombe, *supra* note 24.
[82] Köller et al., *supra* note 25.
[83] Richard A. Lewis, *Chronic inflammatory demyelinating polyneuropathy*, 30 CURRENT OPINION NEUROLOGY 1 (2017), filed as "Pet. Ex. 70."

common host factors that influence susceptibility to these disorders. Pet. Ex. 60 at 1.[84] While GBS and CIDP have some similarities, there are also clear differences:

> Antiganglioside antibodies are frequently detected in GBS, but are generally absent in CIDP. Preceding infections are less frequently reported in CIDP, but infections during the course of CIDP may clearly worsen symptoms . . . most CIDP patients improve after steroids, whereas GBS patients do not. On the other hand, most GBS and CIDP patients improve after IVIg or plasma exchange.

Pet Ex. 60 at 2.[85] Most patients fit the diagnostic criteria of either GBS or CIDP, and though extremely uncommon, a patient may have separate episodes of GBS and CIDP and must be diagnosed and treated accordingly. *Id.*; *see also* Pet. Ex. 56.[86]

2. Determination of Diagnosis

The petition filed in this matter alleges and Dr. Dahlgren initially opined that petitioner suffered from GBS as a result of a September 27, 2016 flu vaccination. *See* Petition, ECF No. 1; Pet. Ex. 26. In his three subsequent reports, Dr. Dahlgren opined that petitioner suffered from both GBS and CIDP, with the only difference between the two diseases being chronicity. Pet. Ex. 55; Pet. Ex. 64; Pet. Ex. 65. It bears repeating that petitioner alleged that he suffered GBS as a result of his Fluzone vaccination and never amended his petition to include CIDP, even though Dr. Dahlgren ultimately opined that petitioner suffered both GBS and CIDP. Drs. Jamieson and He disagreed, maintaining that GBS and CIDP are two distinct diseases, and one cannot be substituted for the other. Resp. Ex. A; Resp. Ex. C; Resp. Ex. D. Dr. Jamieson opined that petitioner suffered from CIDP. Resp. Ex. A; Resp. Ex. C.

Petitioner's treating neurologist ultimately diagnosed him with CIDP due to the relapsing and remitting nature of his condition and his response to IVIG and steroids. Pet. Ex. 11 at 11, 15, 21; Pet. Ex. 15 at 73; Pet. Ex. 53 at 747, 756. Petitioner died on November 23, 2019, and his cause of death was listed as respiratory failure, CIDP, malnutrition, severe deconditioning, atrial fibrillation, neurogenic bladder, and anemia. Pet. Ex. 49.

Treating doctors' views about the appropriate diagnosis are often persuasive because the doctors have direct experience with the patient they are diagnosing. *See McCulloch v. Sec'y of Health & Human Servs.*, No. 09-293V, 2015 WL 3640610, at *20 (Fed. Cl. Spec. Mstr. May 22, 2015). Based on the history of petitioner's condition, the onset and progression of symptoms from the end of December 2016 through February 2017, his response to steroids and later to IVIG, the relapsing and remitting nature of his symptoms, and the opinion of his treating neurologist, petitioner fit the criteria for a diagnosis of CIDP, not GBS which is a monophasic disease with rapid onset that reaches nadir than resolves.

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[84] Kuitwaard et al., *supra* note 27.
[85] *Id.*
[86] Abnormalities of T cells, antibodies, and gene expression have been reported in the peripheral blood of subjects with GBS and CIDP. Although the two have similar pathological features, the course of the diseases differ: "it would seem very likely that subjects with GBS would differ from subjects with CIDP in their ability to regulate immune attack on peripheral nerve, with GBS being an acute self-limited disease and CIDP an ongoing disease." Blum & McCombe, *supra* note 24 at 1, 10.

B. *Althen* Prongs

1. Petitioner Failed to Show an Appropriate Temporal Relationship Between His Receipt of an Influenza Vaccine and His Development of CIDP.

The Vaccine Injury Table lists GBS as an injury resulting from the administration of the flu vaccine when the first symptom or manifestation of onset appears within between three and forty-two days, and specifically "not less than 3 days and not more than 42 days." *See* 42 C.F.R. § 100.3(a)(XIV)(D). Petitioner reported onset of symptoms over three months, or at the earliest 94 days, after his receipt of the September 27, 2016 flu vaccine. Therefore, an important issue in this case is onset, and whether three months or more after flu vaccination is a medically acceptable timeframe for the onset of CIDP following a flu vaccine. *Althen* Prong III requires petitioner to both "establish the time frame for which it is medically acceptable to infer causation," and show that the onset of his injury occurred during that causation period. *Shapiro v. Sec'y of Health & Human Servs.*, 105 Fed. Cl. 353, 360 (2012), *aff'd*, 503 F. App'x 952 (Fed. Cir. 2013).

Dr. Dahlgren acknowledged that onset was more than three months following petitioner's receipt of the subject flu vaccine, but he argued that petitioner had memory problems, the 42 days used in vaccine cases is an arbitrary number, and the literature he presented supports onset beyond 42 days following flu vaccine. *See* Pet. Ex. 26.

a. Onset of Petitioner's Neurological Problems

Dr. Dahlgren suggested that even though petitioner presented to his physician 12 weeks after vaccination, it is unclear when his symptoms started because of his memory problems and because he reported that his symptoms started "several" days before his presentation but failed to report that he suffered from flu-like symptoms from October 24 to 27, 2015. Pet. Ex. 55 at 1-2. Dr. Dahlgren submitted that the foregoing was indictive of his memory problems, and the date of onset of his weakness should therefore "not be considered as a definitive line disqualification to a conclusion that the flu vaccine was the triggering event" for CIDP. *Id*. at 2.

However, petitioner's wife regularly accompanied him to his medical visits and supplemented his history as evidenced in the medical records. *See* Pet. Ex. 5 at 41, 64; Pet. Ex. 10 at 2; Pet. Ex. 11 at 2. She was present when petitioner presented to the ER reporting onset of numbness "several days" prior, and when he reported onset of tingling in all limbs around December 28, 2016, over the two days he was off from work. Pet. Ex. 5 at 41; Pet. Ex. 11 at 2. She showed the doctor a video of petitioner moving his fingers while sitting still from April 2016 and questioned whether it was a clue to some neurological condition. Pet. Ex. 11 at 2. She did not correct the onset of symptoms he reported. Therefore, an onset around December 28, 2016 is consistent with the evidence.

b. Expected Interval Between Vaccination and Onset

Dr. Dahlgren defined "latency" as the duration of time between the vaccination and the onset of symptoms and argued that the "42 day latency is arbitrary and fails to consider all the evidence. Some vaccine induced autoimmune disease cases have their onset within 42 days, **but**

not all." Pet. Ex. 26 at 3 (emphasis in original). Citing to medical literature that he submitted supported onset occurring later than 42 days, Dr. Dahlgren concluded that the literature supports his opinion that the 42-day period for onset "should not be applied as a hard and fast deadline." *Id*. at 4-12.; Pet. Ex. 55; Pet. Ex.

Dr. He described onset of symptoms three months after vaccination as "a long timeframe that is extremely unlikely caused by any immune responses stimulated by his influenza vaccine." Resp. Ex. D at 8. Dr. He pointed out that Dr. Dahlgren misused the term "latency" throughout his reports, because the term indicates an established causal relationship, "which is certainly not the case" for the seasonal flu vaccine and GBS or CIDP. *Id*. at 9.

Dr. He explained that the six-week or 42-day temporal relationship between vaccination and GBS in the Table is "very much consistent with the nature of an immune response to vaccination or infection." Resp. Ex. D at 9. In the case of a flu vaccination, antibody-secreting cells peak at seven to eight days after vaccination then rapidly decline, with low numbers of antibody-secreting cells detected 11 days post-vaccination. In less than three weeks, antigen-specific T cells undergo a contraction phase, forming a stable memory T cell pool. Thus, an immune response to a foreign antigen completes its process in about three weeks, and the six-week timeframe covers two cycles of immune responses, which is "undoubtedly more than enough time" for a vaccination-induced immune response to occur, if GBS was in fact caused by flu vaccine. *Id*. Dr. He noted that the increased risk of GBS after the 1976 swine flu vaccination campaign was within six weeks following vaccination, with no increased risk of GBS found beyond six weeks. *Id*.; Resp. Ex. D, Tab 12.[87]

Dr. He then addressed the literature relied on by Dr. Dahlgren, noting that Dr. Dahlgren "mischaracterized, misinterpreted, or made wrong claims based on his cited references." Resp. Ex. D at 19. Dr. He opined that much of the literature Dr. Dahlgren filed was not related to GBS, flu vaccine, or both. Resp. Ex. D at 11-14; Pet. Ex. 45[88]; Pet. Ex. 48[89]; Pet. Ex. 37[90]; Pet. Ex. 28[91]; Pet. Ex. 38[92]; Pet. Ex. 31.[93] Further, none of the literature provided supports that onset could occur more than three months after vaccination as alleged by Dr. Dahlgren. Resp. Ex. D at 16-19. In fact, Dr. He explained that several of the studies filed by Dr. Dahlgren actually support the 42-day onset interval. *Id*. at 17; Pet. Ex. 43[94]; Pet. Ex. 32.[95] According to Dr. He, the onset of autoimmune disease within three to 42 days is not only based on evidence from many studies but is also strongly supported by the natural cycles of immune responses to foreign antigens. Resp. Ex. D at 9, 17. He submitted that *Sejvar* concluded that six weeks "following any identified antecedent event would represent a reasonable period of surveillance, beyond which biological plausibility of an association with an identifiable antigenic stimulus (e.g., infectious illness, vaccination) declines."

[87] Safranek et al., *supra* note 57.
[88] Soriano et al., *supra* note 11.
[89] Watad et al., *supra* note 15.
[90] Tomljenovic et al., *supra* note 16.
[91] Agmon-Levin et al., *supra* note 17.
[92] Liu & Kaplowitz, *supra* note 19.
[93] Colafrancesco et al., *supra* note 18.
[94] Schonberger at al., *supra* note 13.
[95] Dodd et al., *supra* note 20.

Id. at 18; Pet. Ex. 44.[96] Finally, Dr. He noted that latency was not discussed in any of the literature relied on by Dr. Dahlgren. Resp. Ex. D at 17.

Dr. Jamieson did not address the onset issue in detail other than to say that there is no immunological or epidemiological support for a flu vaccine triggering CIDP three months later. She aptly stated that relying on literature discussing other diseases and other vaccines cannot be substituted for a distinct disease and a flu vaccine. Resp. Ex. A at 20-21; Resp. Ex. C at 3.

Dr. Dahlgren is neither a neurologist nor immunologist. His opinions therefore carry less weight than those of Dr. Jamieson and Dr. He. Further, the literature he relied on is unpersuasive for a number of reasons: he conflated latency and onset, suggesting that the cited literature discussed causation when they discussed temporal relationship; he relied on studies involving the swine flu vaccine and GBS, not seasonal influenza vaccine; he conflated the progressive nature of CIDP, which evolves over a period of two months before reaching nadir, with onset being in excess of two months; and he relied on literature related to other vaccines and other autoimmune diseases, which cannot be substituted for flu vaccine and GBS or CIDP. Finally, not one of the articles cited by Dr. Dahlgren supported onset in excess of eight weeks. Petitioner's onset was 94 days, or more than three months after flu vaccination.

Regardless of whether I determined that petitioner suffered from GBS or CIDP, an onset of 94 days or over three months after flu vaccination is wholly unsupported by the medical literature. Past Program decisions have accepted eight weeks as the outer limit for the onset of a non-Table claim of GBS following flu vaccine. *See, e.g., Williams v. Sec'y of Health & Human Servs.*, No. 19-1177V, 2021 WL 815921, at *5 (Fed. Cl. Spec. Mstr. Jan. 19, 2021) (finding over 100 days is not a reasonable timeframe for onset of a flu vaccine/GBS injury); *Chinea v. Sec'y of Health & Human Servs.*, No. 15-095V, 2019 WL 1873322, at *33 (Fed. Cl. Spec. Mstr. Mar. 15, 2019), *review denied*, 144 Fed. Cl. 378 (2019) (finding that the onset of the petitioner's GBS occurred eleven to twelve weeks after her vaccination, which was beyond the six- to eight-week medically appropriate timeframe for the occurrence of vaccine-induced GBS); *Barone v. Sec'y of Health & Human Servs*., No. 11-707V, 2014 WL 6834557, at *13 (Fed. Cl. Spec. Mstr. Nov. 12, 2014) (finding eight weeks (56 days) is the longest reasonable timeframe for a flu vaccine/GBS injury).

Petitioner has not presented any evidence to support the onset of GBS or CIDP 94 days or over three months after a flu vaccine and has therefore failed to sustain his burden under Prong III.

2. Petitioner Has Not Articulated a Sound and Reliable Medical Theory Causally Connecting Influenza Vaccine to GBS or CIDP.

Petitioner's failure to prove Prong III is dispositive. However, the remaining *Althen* Prongs will be addressed briefly, nonetheless.

The first *Althen* prong requires petitioner provide a "reputable medical theory" demonstrating that the vaccines received *can* cause the type of injury alleged. *Pafford*, 451 F.3d at 1355-56 (citation omitted). To satisfy this prong, petitioner's "theory of causation must be

[96] Sejvar et al., *supra* note 22.

supported by a 'reputable medical or scientific explanation.'" *Andreu*, 569 F.3d at 1379 (quoting *Althen*, 418 F.3d at 1278). This theory need only be "legally probable, not medically or scientifically certain." *Id.* at 1380 (emphasis omitted) (quoting *Knudsen*, 35 F.3d at 548). This standard was clarified by the Federal Circuit. *See Boatmon v. Sec'y of Health & Human Servs.*, 941 F.3d at 1351, 1359-60 (Fed. Cir. 2019) (stating that the correct standard for *Althen* prong one is "reputable," and "sound and reliable" not a "lower reasonable standard" (internal quotations omitted)). Nevertheless, "petitioners [must] proffer trustworthy testimony from experts who can find support for their theories in medical literature." *LaLonde*, 746 F.3d at 1341.

To that end, the experts disagree regarding whether GBS and CIDP are the same disease that only differ in chronicity or duration, and Drs. Jamieson and He argue that no studies support the seasonal flu vaccine as a cause of GBS and/or CIDP.

a. Dr. Dahlgren's Opinion

Dr. Dahlgren opined that petitioner developed the "autoimmune neurological condition of GBS and CIDP" as a result of the flu vaccine he received on September 27, 2016. Pet. Ex. 55 at 13. According to Dr. Dahlgren, other than chronicity, GBS and CIDP are the same disease and have the "same mechanism" or "common etiology" that causes autoimmune damage to myelin through the formation of antibodies against myelin "due to the breakdown in self-recognition due to a failure in the body's immune system." *Id*. at 2-3, 5-7; Pet. Ex. 26 at 3. A vaccine purposely stimulates the immune system, and "a rare number of individuals experience an overactive, erroneous complication of an errant immune reaction" based on predisposition due to individual susceptibility. Pet. Ex. 55 at 3. For both GBS and CIDP, the "treatment is the same, IVIG." *Id*. Dr. Dahlgren articulated his "medical theory" as "[q]uite simply, vaccines increase the activity of the immune system, and in some rare cases, an abnormal autoimmune system attacks and damages the myelin around nerves." Pet. Ex. 64 at 3. He added that in the medical records, petitioner's treating doctors referred to petitioner having both GBS and CIDP. Pet. Ex. 55 at 2-5.

Dr. Dahlgren addressed Dr. Jamieson's opinion, noting that she agreed GBS and CIDP share common pathology and treatment but have different presentations and prognoses. Pet. Ex. 64 at 4. He asserted that suggesting that a difference in clinical course makes them fundamentally different diseases "is not correct science. When a disease has a common mechanism and response to treatment and occurring (sic) after a common over aberrant over stimulation of the immune system, they are related entities." *Id*. Dr. Dahlgren added, "the epidemiology has many reasons to fail to support a finding of causation because epidemiology alone cannot prove causation, only additional underlying science can prove causation . . . Epidemiology only addresses association and it takes other pieces of the picture to reach a conclusion about causation." *Id*. at 4-5. Dr. Dahlgren argued that Dr. Jamieson cited only to negative epidemiological studies to refute a relationship between GBS and vaccines, but "[i]n the case of GBS and CIDP the mechanisms and treatment responses are well worked out for GBS and CIDP and clinch the issue of causation. The mechanism of the immune system attacking the myelin in both GBS and CIDP due to an altered immune system is not addressed by Dr. Jamieson." *Id*. at 5. He noted that Dr. Jamieson agreed that IVIG is effective in both diseases, which suggests a common mechanism. *Id*. at 3.

Dr. Dahlgren also noted that several of the articles Dr. He submitted "explain why in a few cases there can be an autoimmune disease such as GBS and CIDP due to a rare defect in the immune system of a few people who receive a vaccination. Pet. Ex. 65 at 4. He added that the Seg*al & Shoenfeld*[97] article discusses molecular mimicry as a mechanism for autoimmunity from vaccines. He opined that molecular mimicry is a well-studied mechanism to explain how GBS and CIDP occur and that they have similar self-antigens to the myelin protective coating of the nerves. *Id*. This is the only time throughout his four reports that Dr. Dahlgren referenced a mechanism by which the flu vaccine could cause GBS and/or CIDP, but he provided no further explanation.

Dr. Dahlgren concluded that the Vaccine Court has already determined that GBS is a compensable injury and since the evidence supports GBS and CIDP being related, CIDP, like GBS, is a complication of vaccine administration and a compensable injury. Pet. Ex. 64 at 3-5.

b. Drs. Jamieson and He's Opinions

Drs. Jamieson and He disagree with Dr. Dahlgren's assertion that GBS and CIDP are the same disease. The two diseases do not have the same pathophysiology and are not treated the same way, so one therefore cannot be substituted for the other. Drs. Jamieson and He further disagree that there is any support for the seasonal flu vaccine causing GBS or CIDP, noting that the literature referring to vaccine-related GBS is specific to the 1976 and 2009 H1N1 vaccines, not seasonal flu vaccines. Resp. Ex. A; Resp. Ex. C; Resp. Ex. D.

Dr. Jamieson distinguished the two diseases, explaining that CIDP develops slowly over approximately eight weeks, with the initial symptoms taking at least two months to reach nadir. Resp. Ex. A at 12-13. While CIDP may be relapsing and remitting, GBS has a rapid onset, reaches nadir within four weeks of onset, and is monophasic though it may have some residual symptoms. *Id*. The clinical presentations are also different. GBS presents with numbness, paresthesia, and limb pain, and the main feature is bilateral and relatively symmetric weakness of the limbs that progresses over 12 hours to 28 days before reaching nadir. *Id*. at 13. Most GBS patients have a full recovery over weeks or months with physical and occupational therapy, and recurrence is exceedingly rare. *Id*. at 14. CIDP patients present with gradually worsening symmetrical numbness and tingling with motor weakness in the hands, feet, upper arms, and thighs, sometimes with extremity pain and abnormal sensation. There are obvious motor deficits, less prominent sensory loss, and decreased or absent reflexes. *Id*. at 14. The immunologic triggers of GBS and CIDP are also different, with CIDP less frequently associated with a preceding event or disease than GBS. *Id*. at 15. Finally, CIDP has not been associated with any vaccine. *Id*. at 18-20.

Dr. Jamieson agreed that IVIG is used to treat both GBS and CIDP but explained that "[t]reatment of two diseases with a commonly used immunotherapy does not imply an overlapping pathophysiological mechanism," and IVIG is used as treatment for numerous unrelated autoimmune diseases. Resp. Ex. C at 2. She argued that, if response to treatment defined pathophysiological mechanism, then the fact that CIDP responds to steroids while GBS does not mitigates any commonality between the two. *Id*.

[97] Yahel Segal & Yehuda Shoenfeld, *Vaccine-induced autoimmunity: the role of molecular mimicry and immune crossreaction*, 14 CELLULAR & MOLECULAR IMMUNOLOGY 1 (2018), filed as "Pet. Ex. 71."

Dr. He also described GBS as an inflammatory polyneuropathy characterized by acute onset, rapid progression, symmetrical weakness, and hyporeflexia or areflexia. Resp. Ex. D at 3. He noted GBS is post-infectious, with two thirds of patients reporting either respiratory or gastrointestinal tract infection before onset. *Id*. at 4. Although not firmly established, cross-reactive autoantibodies may play a role in the immunopathogenesis of GBS, attacking peripheral nerves and causing demyelination and axonal damage. *Id*.

Dr. He described CIDP as an acquired, immune-mediated neuropathy affecting the peripheral nerves and nerve, roots characterized by a relapsing and remitting or a progressive course, glucocorticoid responsiveness, and electrodiagnostic or pathologic features of demyelination. Resp. Ex. D at 5. The cause and specific immunologic triggers are largely unclear, and no evidence between vaccination and development of CIDP beyond temporal relationship has been provided in any report. *Id*.

Dr. He argued that although it is generally accepted that H1N1 pandemic influenza vaccines were associated with GBS, the same is not true for seasonal influenza vaccines. Resp. Ex. D at 4-5. He addressed Dr. Dahlgren's opinions and the seven papers he relied upon, summarizing that none of them provided evidence of a causal link between seasonal influenza vaccines and GBS. Specifically, he noted that while two of the papers reported an increased risk of GBS in populations that received pandemic swine flu influenza vaccines, the other five showed that there was no statistically significant excess risk of GBS after seasonal flu vaccination, and no causal relationship between the two. Resp. Ex. D at 6-8; Pet. Ex. 30[98]; Pet. Ex. 34[99]; Pet. Ex. 35[100]; Pet. Ex. 36[101]; Pet. Ex. 39; Pet. Ex. 41[102]; Pet. Ex. 42.[103] Importantly, there is no report of GBS caused by the Fluzone vaccine. Resp. Ex. D at 8.

Prior cases in the Program have addressed whether GBS and CIDP are distinct diseases or a continuum of the same disease, referring to the two as "related" peripheral neuropathies with a number of overlapping symptoms that "may" share a common pathogenesis. *See Strong v. Sec'y of Health & Human Servs*., No. 15-1108V, 2018 WL 1125666 (Fed. Cl. Spec. Mstr. Jan. 12, 2018) (referencing the "large and persuasive" body of evidence reliably connecting flu vaccine and GBS and noting the common symptoms and pathogenesis of CIDP and GBS); *Daily v. Sec'y of Health & Human Servs*., 2011 WL 2174535 (Fed. Cl. Spec. Mstr. May 11, 2011) (finding that a theory of CIDP caused by molecular mimicry is plausible based on the premises that flu vaccination can cause GBS, molecular mimicry can cause GBS, and there is a biological basis for similarity between GBS and CIDP). The agreement in these cases, however, is that petitioner can satisfy *Althen* Prong I by relying on the theory of molecular mimicry.

In this case, Dr. Dahlgren's opinions are rooted in the fact that GBS following flu vaccine is a Table injury. However, CIDP is not. For GBS to be considered a Table injury, onset must be within 3 to 42 days of receipt of the flu vaccine. Here, petitioner's onset was at least three months

[98] Martín Arias et al., *supra* note 48.
[99] Haber et al., *supra* note 49.
[100] Haber et al., *supra* note 50.
[101] Hurwitz et al., *supra* note 51.
[102] Marks & Halpin, *supra* note 12.
[103] Salmon et al., *supra* note 53.

after vaccination, and further, he was diagnosed with CIDP. Therefore, this case is an off-Table claim by both timing and injury and requires a sound and reliable theory to sustain petitioner's burden under *Althen* Prong I.

Although petitioner is not required to provide proof of a specific biological mechanism, simply referencing an article that mentions molecular mimicry as a potential mechanism for vaccine-related GBS, and by extension CIDP, without more is insufficient to satisfy petitioner's burden. *See Knudsen ex rel. Knudsen v. Sec'y of Health & Human Servs.*, 35 F. 3d 543, 549 (Fed. Cir. 1994). Molecular mimicry is accepted in the Program as a biological mechanism, but there must be some evidence that persuasively shows that a portion of a vaccine resembles a portion of human tissue which contributes to causing the disease, and that the immune system will respond to the relevant amino acid sequence.[104] *See Tullio v. Sec'y of Health and Human Servs.,* No. 15-51V, 2019 WL 7580149 at *12-14 (Fed. Cl. Spec. Mstr. Dec. 19, 2019), *mot. for rev. denied*, 149 Fed. Cl. 448 (2020); *See, e.g.*, *Stricker v. Sec'y of Health & Human Servs.*, 170 Fed. Cl. 701, 720-21 (2024); *Duncan v. Sec'y of Health & Human Servs.*, 153 Fed. Cl. 642, 661 (2021) (finding the special master did not err in rejecting a bare assertion of molecular mimicry and noting "there is an important difference between the general theory of molecular mimicry and the more specific theory that the vaccine . . . [at issue] is capable of triggering an autoimmune response that culminates in the [petitioner's injury]"); *Caredio v. Sec'y of Health & Human Servs.*, No. 17-79V, 2021 WL 6058835, at *11 (Fed. Cl. Spec. Mstr. Dec. 3, 2021) (indicating that a special master did not err in requiring more than homology and citing *Tullio*); *Yalacki v. Sec'y of Health & Human Servs.*, 146 Fed. Cl. 80, 91-92 (2019) (ruling that the special master did not err in looking for reliable evidence to support molecular mimicry as a theory); *but see Patton v. Sec'y of Health & Human Servs.*, 157 Fed. Cl. 159, 169 (2021) (finding that a special master erred in requiring petitioner submit a study to establish medical theory causally connecting flu vaccine to brachial neuritis).

Dr. Dahlgren is neither a neurologist nor an immunologist. His opinions speak to generalities but fail to explain what in the Fluzone vaccine was similar to or shared homology with host antigens to cause CIDP. In Dr. Dahlgren's own words, his "medical theory" was "[q]uite simply, vaccines increase the activity of the immune system, and in some rare cases, an abnormal autoimmune system attacks and damages the myelin around the nerves." Pet. Ex. 64 at 3. While this may be true, CIDP has no known cause, and Dr. Dalhgren simply referenced the term molecular mimicry in his fourth report, arguing that flu vaccine can cause GBS and CIDP due to the similarity of the two diseases, but failed to connect the flu vaccine to either GBS or CIDP. Nothing in Dr. Dahlgren's reports nor the literature cited explains or references what antigens or structures in the Fluzone vaccine share homology with possible host antigens and how those antigens react to provide a causal connection between GBS or CIDP and seasonal flu vaccine over three months after vaccination. *See Dennington v. Sec'y of Health and Human Servs*., 167 Fed. Cl. 640 (2023) *appeal withdrawn*, No. 2024-1214 (Fed. Cir. Mar. 25, 2024) (criticizing the lack of specificity in petitioner's expert report, suggesting that one could take the expert's causation theory and substitute any table vaccine and any autoimmune disorder with no change to the discussion – "That is how general his molecular mimicry theory is—it does not matter which vaccine and which autoimmune disorder are plugged in. But *Althen* prong one requires more.").

[104] The term generally used to describe this concept in molecular mimicry is homology, or "structural similarity due to descent from a common form." *Homology*, DORLAND'S 858.

It is well-established that the mere mention of molecular mimicry is insufficient to satisfy Prong I. Accordingly, petitioner has failed to sustain his burden under *Althen* Prong I.

3. Petitioner Failed to Demonstrate a Logical Sequence of Cause and Effect.

The second *Althen* prong requires proof of a "logical sequence of cause and effect." *Capizzano*, 440 F.3d at 1326 (quoting *Althen*, 418 F.3d at 1278). In other words, even if the vaccinations can cause the injury, petitioner must show "that it did so in [this] particular case." *Hodges v. Sec'y of Health & Human Servs.*, 9 F.3d 958, 962 n.4 (Fed. Cir. 1993) (citation omitted). "A reputable medical or scientific explanation must support this logical sequence of cause and effect," *id.* at 961 (citation omitted), and "treating physicians are likely to be in the best position to determine whether a logical sequence of cause and effect show[s] that the vaccination was the reason for the injury," *Paluck*, 786 F.3d at 1385 (quoting *Andreu*, 569 F.3d at 1375). Petitioner is not, however, required "to eliminate alternative causes as part of establishing [their] prima facie case." *Doe v. Sec'y of Health & Human Servs.*, 601 F.3d 1349, 1357-58 (Fed. Cir. 2010); *see Walther v. Sec'y of Health & Human Servs.*, 485 F.3d 1146, 1152 (Fed. Cir. 2007) (holding that a "petitioner does not bear the burden of eliminating alternative independent potential causes").

Dr. Dahlgren provided little by way of a medical theory beyond stating, "vaccines increase the activity of the immune system, and in some rare cases, an abnormal autoimmune system attacks and damages the myelin around the nerves," which was insufficient to satisfy *Althen* Prong I. Pet. Ex. 64 at 3. He argued that petitioner had GBS and CIDP because his physicians included both in the record and opined that the only difference between the two diseases is chronicity. Pet. Ex. 55 at 12-13. Dr. Jamieson disagreed, opining petitioner was inappropriately diagnosed with cervical myelopathy and underwent an unnecessary cervical decompression, and he never had GBS, even when he was initially symptomatic. Rather, he had CIDP with a relapsing and remitting course over a three-year period. Resp. Ex. A at 12-13.

Petitioner was ultimately diagnosed with CIDP, and he experienced relapsing and remitting symptoms from onset in December 2016 until his death in November 2019. He also suffered from a host of other medical conditions that required intervention during that period. *See* Pet. Ex. 22 at 1, 24-25; Pet. Ex. 52 at 2159; Pet. Ex. 53 at 257-58, 747, 756; Pet. Ex. 54 at 216-17. When petitioner passed away, his cause of death was respiratory failure and CIDP, along with several other comorbidities. Pet. Ex. 49.

As discussed in detail above, petitioner failed to establish that the Fluzone vaccine he received caused his CIDP over three months after vaccination. It is therefore axiomatic that he would fail to prove a logical sequence of cause and effect between his Fluzone vaccination on September 27, 2016 and his onset of symptoms around December 28, 2016. The preponderant evidence supports that petitioner suffered from CIDP based on his clinical presentation, his responsiveness to steroids and IVIG, and the timing of his onset and the subsequent relapsing and remitting progression of his condition over the next three years until his death. Because petitioner failed to provide a reliable theory for how the Fluzone vaccine could cause CIDP more than three months after vaccination, he likewise failed to provide preponderant evidence of a logical connection between the flu vaccine he received on September 27, 2016, and the onset of CIDP more than three months later.

Accordingly, petitioner has failed to sustain his burden under Prong II.

VII. Conclusion

Upon careful evaluation of all the evidence submitted in this matter—the medical records, petitioner's affidavit, and the experts' opinions and medical literature—I find that petitioner has failed to show he is entitled to compensation under the Vaccine Act. **The matter is hereby dismissed, and the Clerk shall enter judgment accordingly.**[105]

IT IS SO ORDERED.

s/ Mindy Michaels Roth
Mindy Michaels Roth
Special Master

[105] Pursuant to Vaccine Rule 11(a), entry of judgment can be expedited by each party filing a notice renouncing the right to seek review.